Journal of Neurosurgery

MARQUETTE UNIVERSITY
MAY 1 1958
Medical-Dental Library

JULY 1957

VOLUME XIV JULY 1957 NUMBER 4

JOURNAL OF NEUROSURGERY

[*J. Neurosurg.*]

MANAGING EDITOR

LOUISE EISENHARDT, 333 Cedar Street, New Haven 11. Conn.

EDITORIAL BOARD

PAUL C. BUCY, Chicago, *Chairman*

FRANC D. INGRAHAM, Boston
BRONSON S. RAY, New York
DAVID L. REEVES, Santa Barbara
BARNES WOODHALL, Durham

ADVISORY BOARD

A. ASENJO, Santiago de Chile
WINCHELL McK. CRAIG, Rochester, Minn.
GILBERT HORRAX, Boston
GEOFFREY JEFFERSON, Manchester, Eng.
KENNETH G. McKENZIE, Toronto
W. JASON MIXTER, Boston
HOWARD C. NAFFZIGER, San Francisco
HERBERT OLIVECRONA, Stockholm
WILDER PENFIELD, Montreal
D. PETIT-DUTAILLIS, Paris
ERNEST SACHS, New Haven
R. GLEN SPURLING, Louisville

PUBLISHED BI-MONTHLY

FOR THE HARVEY CUSHING SOCIETY BY

CHARLES C THOMAS, Publisher

301-327 EAST LAWRENCE AVENUE, SPRINGFIELD, ILLINOIS, U.S.A.

Copyright, 1957, by Charles C Thomas, Publisher. Subscription rates: $11.00 a year in the United States, Pan-American Union, and Spain; $11.50 a year in Canada; $12.00 a year in other countries; single copies, $2.00 postpaid. Single copies of Volume VIII, Number 1, January 1951 issue: Symposium on Intracranial Vascular Abnormalities: $3.00 postpaid, if available.

Distributed in the British Commonwealth of Nations by Blackwell Scientific Publications, Ltd., 24-25 Broad Street, Oxford, England, Copyrighted in the British Commonwealth by Blackwell Scientific Publications, Oxford. No part may be duplicated or reproduced without permission of publishers. Subscription price £4.7.6.

Entered as second-class matter March 12, 1944 at the post office at Springfield, Illinois, under the act of March 3, 1879. Additional Entry at the post office at Menasha, Wisconsin.

PRINTED IN THE U.S.A.

Gudebrod OFFERS

CERETHERMIC FINISH

Dri-Pak® sutures with Mintraumatic® Needle, sterile, in dry envelopes.

☆ A Complete Line of Silk or Cotton Sutures

☆ The Most Convenient and Most Modern Packaging

Dri-Pak® sutures, silk or cotton, sterile, in tubes.

Add to these the full Gudebrod line of sterile and non-sterile sutures—plus Gudebrod specialties—and every operating room need for non-absorbable sutures is fulfilled.

Dri-Pak® sutures, Detachable® cut lengths or ligature reels, sterile, on reels, in dry envelopes.

Champion Silk sutures or Cotton sutures, on Rubber tubes or spools, non-sterile.

☆ Superior Handling Qualities for the Surgeon

☆ Simpler Procedures for the Operating Room Supervisor

Gudebrod BROS. SILK CO., INC.

Surgical Division: 225 West 34th St., New York 1, N. Y.

Executive Offices: 12 South 12th St., Philadelphia 7, Pa.

CHICAGO • BOSTON • LOS ANGELES

Confinia Neurologica

Borderland of Neurology — Grenzgebiete der Neurologie
Les Confins de la Neurologie

EDITORIAL BOARD:

L. Alexander, Boston, Mass.; *A. Austregesilo*, Rio de Janeiro; *A. Barré*, Strasbourg; *M. Bender*, New York; *G. Bodechtel*, Munich; *L. van Bogaert*, Anvers; *T. Broman*, Göteborg; *A. Brückner*, Basel; *H. Brunner*, Newark, N.J.; *H. Cairns*, London; *P. Cokkalis*, Athens; *P. Delmas-Marsalet*, Bordeaux; *Th. Démétriades*, Athens; *Temple Fay*, Philadelphia, Pa.; *J. F. Fulton*, New Haven, Conn.; *F. Georgi*, Basel; *L. Halpern*, Jerusalem; *A. Hautant*, Paris; *G. Holmes*, London; *B. Horanyi*, Budapest; *L. van der Horst*, Amsterdam; *J. Igersheimer*, New York; *W. Klestadt*, New York; *A. Knapp*, New York; *K. Krabbe*, Copenhagen; *H. Kuhlenbeck*, Philadelphia, Pa.; *F. Leiri*, Helsinki; *R. Leriche*, Strasbourg; *J. Lhermitte*, Paris; *R. Lorente de Nó*, New York; *E. Ludwig*, Basel; *D. J. McCarthy*, Philadelphia, Pa.; *G. H. Monrad-Krohn*, Oslo; *F. Naville*, Geneva; *C. Nylén*, Uppsala; *I. Olninck*, New York; *K. Orzechowski*, Warsaw; *T. J. Putnam*, Los Angeles, Calif.; *E. Rutishauser*, Geneva; *E. Sachs*, New Haven, Conn.; *A. Schüller*, Melbourne; *J. Staehelin*, Basel; *R. Wartenberg*, San Francisco, Calif.; *P. H. Wilcox*, Traverse City, Mich.; *H. T. Wycis*, Philadelphia, Pa.

REDACTORES:

A. Franceschetti, Geneva *A. R. Jentzer*, Geneva *L. Rüedi*, Zurich
E. B. Streiff, Lausanne

Editor: E. A. SPIEGEL, Philadelphia, Pa.

"Confinia Neurologica" intends to maintain the mutual relationship of neurology and surgery, oto-ophthalmology, syphilidology, endocrinology and radiology, giving due consideration also to the theoretical foundations. Complete original articles, reviewing articles, society transactions, and book reviews are published. The articles may appear in either English, French or German; the original papers are summarized in the 3 languages. The reviews of the literature (about 8-10 yearly) present a survey of each of the above disciplines, so that the collected issues will represent a yearbook on the progress in the borderland of neurology.

---

1 volume of 6 parts is published yearly. Subscription price U.S. $12.00 per volume

---

BASEL 11 (Switzerland) S. KARGER NEW YORK
For U.S.A.: Albert J. Phiebig, P.O. Box 352, White Plains, N.Y.

storz
Surgical Instruments

NEW!

Tissue and Dressing FORCEPS on Spring Handle

For Plastic, Thoracic, or Neurosurgery

Six forceps of improved design to obtain a better functioning spring handle forceps, which can be held far back on the handle but obtain a firmer grip at the distal end, as there is greatly increased leverage.

All the forceps have crossed branches, they permit a delicate but firm grasp.

#G-1956 is recommended for holding the dentate ligament in cordotomies.

The crossed branches necessitate less space for manipulation.

The fulcrum near the tip prevents overriding of jaws. The lock on #G-1950, #G-1956 and #G-1962 is released by applying additional pressure, which does not cause movement at the tip.

G-1950 Forceps, Tissue, Cross Action: 1 × 2 teeth, 9″, with lock $21.50

G-1952 Forceps, Tissue, Cross Action: 1 × 2 teeth, 9″, without lock 17.50

G-1956 Forceps, Dressing, Cross Action: serrated jaws, 9″, with lock 21.50

G-1958 Forceps, Dressing, Cross Action: serrated jaws, 9″, without lock 17.50

G-1962 Forceps, Tissue, Cross Action: BROWN type jaws, 2 rows of fine teeth, 9″, with lock 22.75

G-1964 Forceps, Tissue, Cross Action: BROWN type jaws, 2 rows of fine teeth, 9″, without lock 18.75

●

STORZ INSTRUMENT CO., 4570 Audubon Avenue, St. Louis 10, Missouri

Second Edition

THE HEAD AND NECK IN ROENTGEN DIAGNOSIS

By

EUGENE P. PENDERGRASS, M.D., *Professor of Radiology and Chairman of the Department of Radiology, University of Pennsylvania*

J. PARSONS SCHAEFFER, M.D., Ph.D., *Professor of Anatomy and Director of the Daniel Baugh Institute of Anatomy, Emeritus, Jefferson Medical College*

PHILIP J. HODES, M.D., *Professor of Radiology, University of Pennsylvania*

IN TWO LARGE VOLUMES

Complete index for both volumes in each volume

In part *rewritten* and *rearranged,* at places *greatly extended,* and new sections added.

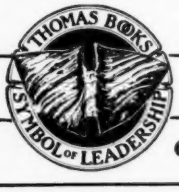
NEW TECHNICS — NEW INTERPRETATIONS
NEW DESCRIPTIONS — NEW ILLUSTRATIONS

THE MOST EXPERT KNOWLEDGE ABOUT THE ROENTGEN DIAGNOSIS OF THE **DISEASES, INJURIES,** AND **ANATOMICAL VARIATIONS** OF THE HEAD AND NECK:

- Normal and abnormal conditions
- Conformations
- Fundamental and Variational anatomy
- Developmental variations
- Physical signs and symptoms
- Functional and pathological observations of the head and neck region exclusively

2403 illustrations, many new, illuminate the text

ILLUSTRATIONS: The 2403 illustrations deserve special comment for their **newness, clarity,** and **usefulness.** They are not only superior in concept and scope but most all are new and original. Many reveal new facts not heretofore included in the literature. Many of these illustrations are concerned with basic technical procedures. All are carefully prepared and unusual anatomical illustrations which make new aspects of anatomy available. The greatest care was exercised to correlate anatomical structures with the shadows seen on roentgenograms.



1880 pages (7 x 10) **2403 illustrations** **$37.50**

CHARLES C THOMAS • PUBLISHER • SPRINGFIELD • ILLINOIS

This myelogram, made with the spot-film device, shows a herniated sixth cervical intervertebral disk. A concave filling defect of the *Pantopaque* column in the subarachnoid space is present, extending from the midportion of the body of the sixth cervical vertebra to the lower margin of the seventh cervical vertebra on the left side. The sheath of the nerve root between the sixth and seventh cervical vertebrae is obliterated, and the root sheath between the seventh cervical and first thoracic vertebrae is deformed. The cervical spinal cord is displaced slightly toward the right.

"PANTOPAQUE" *is the registered trademark under which all leading x-ray dealers supply the compound ethyl iodophenylundecylate, which is synthesized by the Research Laboratories of Eastman Kodak Company and prepared as the myelographic contrast medium Iodophenylate Injection, U.S.P., by Lafayette Pharmacal Company. The trademark serves to indicate to the radiologist continuity of experience in the manufacture of this medium.*

Announcing . . .

THE EARLY DIAGNOSIS AND TREATMENT OF ACOUSTIC NERVE TUMORS

By

J. LAWRENCE POOL, M.D.
Professor of Neurological Surgery, Columbia University
Director of Service of Neurological Surgery
The Neurological Institute
Presbyterian Hospital, New York, New York

and

ARTHUR A. PAVA, M.D.
Assistant Resident Neurological Surgeon
The Neurological Institute
Presbyterian Hospital, New York, New York

A Monograph in **American Lectures in Surgery** *edited by* MICHAEL E. DE BAKEY, R. GLEN SPURLING, *and* BARNES WOODHALL.

Assembles under one cover the experiences of others as well as the authors, dealing with the SURGICAL MANAGEMENT and the RESULTS of total and subtotal removal of these tumors.

In this brief but comprehensive monograph on the diagnosis and treatment of eighth nerve tumors the authors stress the fact that early diagnosis leads to better chances for total tumor removal with uncomplicated post-operative results. **Clinical and laboratory means of identifying the presence of an acoustic neurinoma are presented and differential diagnosis is discussed.** Reference is also made to the fact that occasionally two tumors may occur in a given patient such as bilateral acoustic neurinomas, or an eighth nerve neurinoma and an intracranial meningioma elsewhere.

Historical and pathological data are presented together with a detailed description of operative techniques and precautions, including indications for total and subtotal tumor removals.

Altogether this monograph should prove helpful not only to neurologists and neurological surgeons, but to the general practitioner and otolaryngologist.

1957 **162 pages** **49 illustrations**

Sent on approval, $5.50

You'll be interested also in . . .

THERAPEUTIC EXERCISES FOR THE TREATMENT OF THE NEUROLOGICALLY DISABLED

By **HAROLD J. BRENNER, M.S.,** *Assistant Chief, Corrective Therapy, Supervisor of the Neurological Service, Veterans Administration Hospital, Sepulveda, California.* A text for corrective therapists and corrective physical educators. A PRACTICAL and BASIC GUIDE for rehabilitation through exercise. The author interviewed numerous students, therapists and corrective physical education teachers for information that would be most pertinent for their specific needs. **1st Edition published 1957, 84 pages, 19 illustrations, Cloth, $3.50**

BRAIN MECHANISMS AND DRUG ACTION

A Symposium of the Fourth Annual Scientific Meeting of the Houston Neurological Society, compiled and edited by **WILLIAM S. FIELDS, M.D.,** *Professor of Neurology, Baylor University College of Medicine.* The functions of the brain stem reticular formation are summarized and the effects of chlorpromazine, reserpine and of cholinergic and anti-cholinergic agents upon the reticular system are discussed. **1st Edition published 1957, 160 pages, 150 illustrations, Cloth, $4.25**

SPINAL CORD COMPRESSION: MECHANISM OF PARALYSIS AND TREATMENT

By **I. M. TARLOV, M.D.,** *Professor of Neurology and Neurosurgery, New York Medical College, New York, New York.* From on-the-scene-of-accident management to the question of whether and when to operate, based on experimental and clinical evidence. **1st Edition published 1957, 164 pages, 88 illustrations, Cloth, $7.50**

CHARLES C THOMAS • PUBLISHER • SPRINGFIELD • ILLINOIS

For the Neurosurgeon:

the Stryker TURNING FRAME

. . . provides an excellent method of immobilization in treatment of cord injuries.

Stryker SURGICAL AND HOSPITAL EQUIPMENT

Orthopedic Frame Company

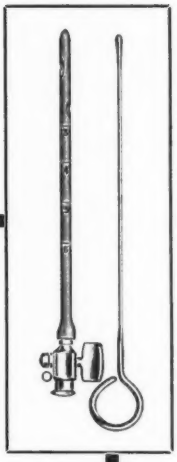
420 ALCOTT STREET • KALAMAZOO, MICHIGAN

Distributed in Canada by: Fisher & Burpe, Ltd., Winnipeg

Exclusive Agent for Export: Schueler & Co., 75 Cliff St., N. Y.

non-rigid
Ventricular Cannula

by Emil Seletz, M.D.



This flexible, radio-opaque rubber cannula has the proportions of standard metal cannula (10F, 9cm.), calibrated in centimeters; rubber is covered by special lacquer for smoother passage through brain tissue. Lightweight metal hub fitted with airtight, watertight stopcock. Tube has removable stilet.

Cannula may be left in place during x-ray and surgery—can serve as safety valve in ventricle 24 to 36 hours following craniotomy . . . has served as a life-saving measure for gradual decompression in acute intracranial pressure in comatose patients. Air release after ventriculography requires mere opening of stopcock.

NL-2130 . . . . . Each, $15.00

Armamentarium

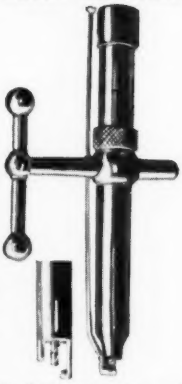
V. MUELLER & CO.
330 South Honore Street
Chicago 12, Illinois
Dallas • Houston • Los Angeles • Rochester, Minn.

By Nudell . . .

A NEW INSTRUMENT FOR NEUROSURGEONS

CRANIUM PLATE-SEAT CUTTER

Neatly grooves cranium to flush-fit polyethylene or metal plates.



This instrument quickly cuts a smooth, even, recessed seat which adds to the rigidity of a polyethylene plate, as well as flushing it with the top of the skull. Depth of cut is adjustable. Has a rotary cutting blade and water-cooling tube. Available with direct drive motor or adapted for flexible shaft. Instrument is chrome plated and may be autoclaved.

Write for literature on Nudell's Skull Templates and other specialities for Neurosurgeons.

UZA NUDELL
Technician to Neurosurgeons
125 W. 45TH STREET • NEW YORK 36, N.Y.

BRAIN MECHANISMS IN DIACHROME

by

WENDELL J. S. KRIEG
PROFESSOR OF ANATOMY,
Northwestern University Medical School.

A brief, graphic summary of neuroanatomy, featuring the prize-winning reconstruction of the human brain in full color, on 12 pages of transparent acetate. 188 pages. 57 figures. $7.00, postpaid, when ordered directly from

BRAIN BOOKS
BOX NINE EVANSTON, ILL.

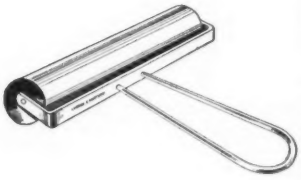
Cranioplastic

RESINOUS MATERIAL FOR REPAIRING CRANIAL DEFECTS

A method of rapidly repairing cranial defects using a resin material containing Methyl-methacrylate which is molded into the cranial defect at the time of the original operation. This highly refined plastic has been used since 1947 and to date it has not been necessary to remove a single plate.

N-196 CRANIOPLASTIC KIT

Contains 3 packets of powder (sterile), 3 ampules of liquid (sterile) and 3 matrix bags which is sufficient material for the repair of three ordinary cranial defects.



N-197 SPENCE CRANIOPLASTIC ROLLER

A stainless steel roller which is of great help in rolling the plastic mass to the desired thickness before fitting to the cranial defect.

N-198 CARBORUNDUM WHEEL

Used with bone saw or drill to bevel edges and smooth rough spots. Can be autoclaved.

Ref.: Spence, William T.: Form Fitting Plastic Cranioplasty. Journal of Neurosurgery. May, 1954.

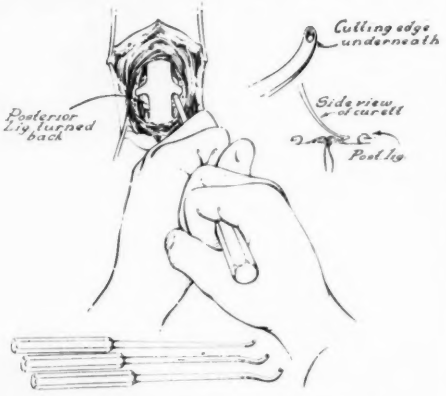
ESTABLISHED 1838

Codman & Shurtleff, Inc.

MAKERS OF SURGEONS' INSTRUMENTS

104 BROOKLINE AVENUE BOSTON 15, MASSACHUSETTS

MAYFIELD CERVICAL CURETTE



KEES SURGICAL SPECIALTY COMPANY

117 W. MAIN STREET ALEXANDRIA, KENTUCKY

An Authorized Binding for

JOURNAL OF NEUROSURGERY

Arrangements have been made for subscribers to have their journals bound into distinctively designed books.

Six issues (bi-monthly) bound by the year, in best grade cocoa washable buckram, imprinted with your name on cover, cost but $3.95 per volume.

Bound journals serve as an immediate reference for research and information. Properly displayed, they create a psychological impact on the patient, implying the time and effort spent to keep up-to-date on the most modern techniques and treatment.

Ship journals parcel post. Within thirty days after receipt, bound volumes will be shipped prepaid anywhere in the U.S.A. Full remittance must accompany order.

PUBLISHERS' AUTHORIZED BINDERY SERVICE

(Binders of all Journals)

5811 W. Division Street, Chicago 51, Illinois

SELECTED WRITINGS OF

WALTER E. DANDY

Compiled by

CHARLES E. TROLAND, M.D.

Dual Purpose:

. . . to honor the memory of Doctor Walter E. Dandy

. . . to make available many of his writings not obtainable from other sources

Certainly **all neurosurgeons and neurologists should have this book,** both as a historical volume and because of the ready availability of information.

Some of the writings of this pioneer in neurosurgery have been reviewed as follows:

"Dandy's operative results were remarkable."—*Archives of Neurology and Psychiatry*

". . . we hail with deepest appreciation the appearance of the present volume from the pen of the eminent author, who, in 1918 gave us a method which facilitates the differential diagnosis of deep-seated brain tumors—ventriculography. Dr. Dandy stands out as the only neurosurgeon who has treated as many as 21 cases of tumors of the third ventricle, there being on record only 16 authentic cases observed by as many individual authors." —*The Military Surgeon*

"The clinical histories and operative notes are well arranged, clearly written and instructive . . . the facts of clinical analysis are interesting and useful."—*Canadian Medical Association Journal*

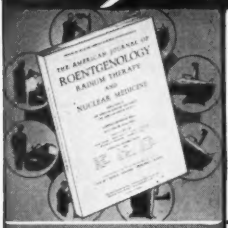
912 pages **183 illustrations**

Cloth, $15.00

CHARLES C THOMAS • PUBLISHER

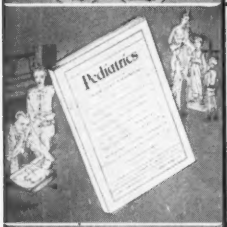
Springfield • Illinois

Six Keys to Success!



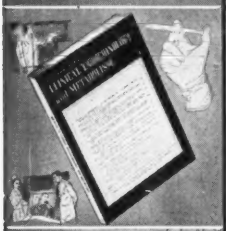
The American Journal of Roentgenology Radium Therapy and Nuclear Medicine

Official Organ—The American Roentgen Ray Society and The American Radium Society. Edited by **Lawrence Reynolds.** Price a year: United States, U. S. Possessions, Pan-American Union and Spain, $15.00; Canada, $16.00; other foreign countries, $17.00. Monthly.



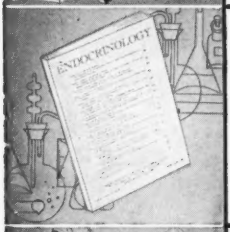
Pediatrics

Official Journal—American Academy of Pediatrics. **Edited by Charles D. May. Price a year: United States, U. S. Possessions, Pan-American Union and Spain, $12.00; Canada, $13.00; other foreign countries, $14.00. Monthly.**



The Journal of Clinical Endocrinology and Metabolism

Official Journal—The Endocrine Society. Edited by **Phebe K. Thompson.** Price a year: United States, U. S. Possessions, Pan-American Union and Spain, $15.00; Canada, $15.50; other foreign countries, $16.00. Monthly.

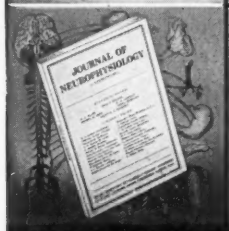


Endocrinology

Official Journal—The Endocrine Society. Edited by **Roy O. Greep.** Price a year: United States, U. S. Possessions, Pan-American Union and Spain, $15.00; Canada, $15.50; other foreign countries, $16.00. Monthly.

Journal of Neurosurgery

Official Journal—The Harvey Cushing Society. Edited by **Louise Eisenhardt.** Price a year: United States, U. S. Possessions, Pan-American Union and Spain, $11.00; Canada, $11.50; other foreign countries. $12.00 Bi-monthly.



Journal of Neurophysiology

Editorial Board: **John F. Fulton, Stephen W. Kuffler, P. D. MacLean, H. W. Magoun, James L. O'Leary, Theodore C. Ruch, Donal Sheehan, Clinton N. Woolsey.** Price a year: United States, U. S. Possessions, Pan-American Union and Spain, $9.50; Canada, $10.00; other foreign countries, $10.50. Bi-monthly.

CHARLES C THOMAS • PUBLISHER • SPRINGFIELD • ILLINOIS

Atlas of TUMORS of the NERVOUS SYSTEM

By

H. M. ZIMMERMAN, M.D.

Chief, Laboratory Division, Montefiore Hospital, and Professor of Pathology, College of Physicians and Surgeons, Columbia University, New York City

MARTIN G. NETSKY, M.D.

Associate Neuropathologist and Associate Attending Physician in Neuropsychiatry, Montefiore Hospital, New York City. Presently Professor of Neuropathology and Associate Professor of Neurology, The Bowman Gray School of Medicine of Wake Forest College, Winston-Salem, North Carolina

LEO M. DAVIDOFF, M.D.

Attending Neurological Surgeon, Montefiore Hospital, and Professor and Chairman, Department of Surgery, Albert Einstein College of Medicine, New York City

WITH an average of more than one color plate per page, this authoritative one-volume atlas depicts both *gross and microscopic features of all tumors of the brain, spinal cord and peripheral and sympathetic nervous systems.* Full attention is devoted, also, to space-occupying lesions such as infectious granulomas and metastatic tumors. The extensive use of color in this beautifully printed book serves two practical purposes: *1,* It enables all who use it to compare their own specimens with the diagnostic illustrations; 2, It becomes an actual and highly desirable substitute for the insufficient quantity of tumor material of the nervous system that is available in most medical centers. In addition to the profuse *diagnostic guidance* this atlas contains, there is full consideration of the biologic behavior, incidence, localization, treatment and prognosis of the neoplasms discussed. The effects of increased intracranial pressure on the brain are covered in detail.

With the growth of neurological surgery has come a complexity of surgical procedures for numerous operations other than brain tumors. That is why this graphic presentation has such great practical appeal to neurosurgeons, surgeons, neurologists, pathologists, orthopedists, radiologists and others whose work touches upon this important branch of medicine.

Partial Contents

Intracranial Tumors. Gliomas, Meningiomas, Neurinomas, Pituitary and Congenital Tumors, Sarcomas, Choroid Plexus Tumors, Metastatic Neoplasms. Granulomas. Effects on Brain from Increased Pressure. **Spinal Tumors:** Gliomas, Meningiomas, Perineurial Fibroblastoma, Congenital Tumors, Metastatic Neoplasms, Effects of Compression of the Spinal Cord.

Tumors of the Peripheral Nerves: Perineurial Fibroblastoma, Neurofibroma and Neurofibromatosis, Neurogenic Sarcoma. **Tumors of Sympathetic Ganglions:** Ganglioneuroma. Neuroblastoma. **Tumors of Paraganglionic Cells. Bibliography. Appendix. Index.**

191 Pages, 7″ × 10″. 277 Illustrations, 233 in Color. 4 Tables. $25.00

(Published November, 1956)

LEA & FEBIGER WASHINGTON SQUARE PHILADELPHIA 6 PA.

An established systemic hemostat

Adrenosem®
SALICYLATE*
(Brand of carbazochrome salicylate)

effective in virtually every operative procedure

The reports of numerous investigators have established the effectiveness of Adrenosem Salicylate in the control of bleeding. It has been used prophylactically and therapeutically in virtually every operative procedure. Following are some of the procedures and conditions where its usefulness has been demonstrated:

Tonsillectomy, adenoidectomy and nasopharynx surgery
Prostatic, bladder and transurethral surgery
Excessive postpartum bleeding and uterine bleeding
Thoracic surgery
Plastic surgery
Gastrointestinal bleeding

ALSO:
Idiopathic purpura
Epistaxis
Retinal hemorrhage
Hematuria
Hemoptysis
Familial telangiectasia
Pulmonary bleeding
Metrorrhagia and menorrhagia

*U.S. Pat. 2581850; 2506294

THE S. E. MASSENGILL COMPANY
Bristol, Tennessee
New York • Kansas City • San Francisco

One of the many uses of **Adrenosem®**
SALICYLATE

Adrenosem Salicylate is useful in every operative procedure where bleeding and oozing are problems. This hemostatic agent controls blood loss by maintaining normal capillary permeability and by promoting the retraction of severed capillary ends.

One clinician states: "At the recommended dosage levels there are no contraindications. Patients treated for more than two years show no toxic effects attributable to the drug."[1]

Adrenosem Salicylate is supplied in

Ampuls
Tablets
and as a Syrup

1. Riddle, A.C., Jr.: Adrenosem Salicylate: A Systemic Hemostat, Oral Surg., Oral Med., Oral Path. 6:617 (June, 1955).

Vaginal hysterectomy.
Without Adrenosem. Note bloody field.

Vaginal hysterectomy.
With Adrenosem. Note dry field.

Write for comprehensive illustrated brochure describing the action and uses of Adrenosem Salicylate.

THE S. E. MASSENGILL COMPANY
Bristol, Tennessee
New York • Kansas City • San Francisco

SPATIAL RELATIONSHIPS BETWEEN THE GLOBUS PALLIDUS AND THE ANTERIOR COMMISSURE*

EDGAR M. HOUSEPIAN, M.D., AND MALCOLM B. CARPENTER, M.D.†

Departments of Neurosurgery and Anatomy, College of Physicians and Surgeons, Columbia University, and the Neurological Institute of Presbyterian Hospital, New York, New York

(Received for publication October 24, 1956)

STEREOTAXIC methods appear to offer the best available means of producing localized lesions in subcortical sites. This technique has the advantage of minimal surgical trauma and incidental tissue destruction. Since the stereotaxic instrument was devised and introduced by Horsley and Clarke,[4] it has proved to be one of the most profitable experimental methods for studying the anatomy and physiology of the brain in animals. The method as used in animal investigation is based upon the principle of substitution of mechanical direction of electrodes, with respect to established planes, for visual direction and the premise that the skull bears a fairly constant relationship to the brain. The greatest experience with this method, to date, has been with experimental animals, particularly the cat and rhesus monkey. These experiences have shown that the method, as commonly employed with experimental animals without roentgenographic control, has a large element of trial and error.[1] No study has been made of the combined use of radiographic techniques and the stereotaxic methods in laboratory animals, but it would seem that considerable increase in accuracy might be obtained.

The hope of Horsley and Clarke that the stereotaxic instrument would prove useful in human neurosurgery was not realized until 1947, when Spiegel *et al.*[6] devised the first human instrument. Because of wide variations in the size and configuration of the human skull, these authors[7] established coordinate systems with respect to structures within the brain, visible in plain roentgenograms (pineal) or identifiable in pneumoencephalogram (i.e., suprapineal recess, posterior commissure and anterior commissure). Narabayashi *et al.*[5] have determined coordinates for the human brain with respect to three intersecting planes: 1) a horizontal plane determined by the inferior ridge of the orbit and both external auditory poles, 2) a vertical plane perpendicular to the horizontal plane and passing through the auditory poles, and 3) a mid-sagittal plane. The point of intersection of these three planes was taken as the zero reference point for calibrations, but final coordinates depended

* Aided by a grant (No. 144) from the National Multiple Sclerosis Society.

† John and Mary R. Markle Scholar in Medical Science.

upon additional pneumoencephalographic information. This latter method is essentially the same as that used in animal investigation, except that roentgenographic assistance is rarely available.

The purpose of the current report was the determination of the spatial relationship between the globus pallidus and the anterior commissure. Since the latter structure often can be localized in a satisfactory pneumoencephalogram and bears an intimate and constant relationship to the globus pallidus, it would seem to be the most reliable reference point upon which to base coordinate systems for stereotaxic approaches to the globus pallidus. A graphic method of representation was devised for determining the general size, shape and disposition of structures within the brain which would appear to facilitate establishment of stereotaxic coordinate systems. By this method data concerning the size, shape and spatial disposition of the globus pallidus with respect to the anterior commissure were obtained.

MATERIAL AND METHODS

The material used in this study consisted of 15 brains obtained from general autopsies. Ages of the patients from whom brains were taken ranged from 32 to 84 years and averaged 60 years. Eight of the patients were males and 7 were females. Death resulted from non-neurologic pathology in all cases, but pre-existing cerebral pathology was found in 3 brains and cerebral arteriosclerosis was marked in 2 other brains. Brains were fixed in 20 per cent formalin for 9–12 days, according to the routine procedure followed in the Division of Neuropathology. One brain was further fixed in 90 per cent alcohol for 14 days. Measurements were taken from 1 additional fresh brain for comparative purposes only. Weights of the brains varied from 1000 to 1525 gm. following removal. None of the brains had generalized or localized edema, gross or localized atrophy, or distortion of midline structures.

The brain stems and cerebella were removed by a transverse cut across the upper mesencephalon. The cerebral hemispheres were cut in 1-cm. transverse sections in a brain-slicing apparatus; 0.5-cm. sections were made when needed. The frontal poles of the brain were placed against a horizontal board and the basal surfaces of the temporal and occipital lobes were pressed against a vertical board. The angle formed by the plane of the basal surfaces of the temporal and occipital lobes and a line projected along the orbital surface of the frontal lobe towards the frontal pole measured approximately 140 degrees (Fig. 1). This angle corresponds closely to the angle formed by lines projected from the lowest point of the middle fossa to the anterior limit of the anterior fossa and to the internal occipital protuberance, forming an angle which can be measured in lateral films of the skull (Fig. 2). Brain slices were thus cut perpendicular to a plane corresponding to that established by the lowest points of the temporal fossae and the internal occipital protuberance.

The center of the anterior commissure was considered the zero reference point. A line passing through the midpoint of the anterior commissure perpendicular to the plane of the brain sections constituted the Z axis. Measurements in millimeters of the rostral and caudal extent of the globus pallidus with respect to the zero point were made along this axis. The X axis was considered as passing from side to side

through the center of the anterior commissure parallel to the plane of the sections. Measurements in millimeters of the medial and lateral limits of the globus pallidus with respect to the zero point were made along this axis. The Y axis consisted of a line passing through the zero point parallel to the plane of section, but perpendicular to the X axis. Measurements along the Y axis were made of the dorsal and ventral extent of the globus pallidus with respect to the zero point.

Brain sections rostral to the anterior commissure were designated AA, BB, CC, etc., while those caudal to it were designated A, B, C, etc. The portion of the globus pallidus on the cut surface of each brain section was outlined as a triangular struc

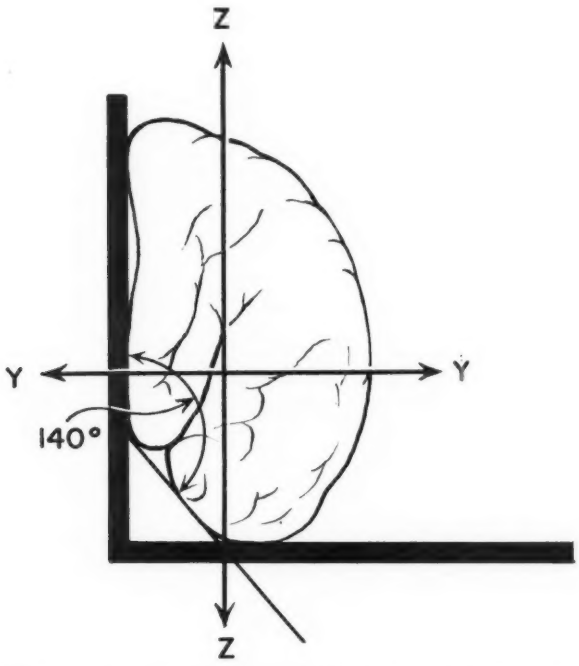


FIG. 1. Drawing showing the position of the cerebral hemispheres in the brain-slicing apparatus. One-cm. transverse sections were cut perpendicular to the Z axis by this method.

ture and each point of the triangle was designated by a number. The position of each of these numbered points in each section containing globus pallidus was determined with respect to the three axes and the zero reference point. Using coordinates thus obtained three graphs of the configuration of the globus pallidus with respect to the axes were drawn for each hemisphere: a lateral projection, a horizontal projection and a frontal projection. In these graphs the plotted points were joined and an attempt was made to smooth the resulting curves. These plots of the three projections of the globus pallidus with respect to the axes and the zero reference point (i.e., center of the anterior commissure) provided data concerning the spatial relationships between the globus pallidus and the anterior commissure.

OBSERVATIONS

The rostrocaudal length of the globus pallidus measured with respect to the Z axis ranged from 19 to 26 mm. and averaged 22 mm. The mean extent of the globus pallidus caudal to the anterior commissure, as measured along the Z axis, was 18 mm., while the pallidum was present rostral to the anterior commissure for a mean distance of 4 mm. with respect to this axis.

The maximum dorsoventral measurements of the globus pallidus with

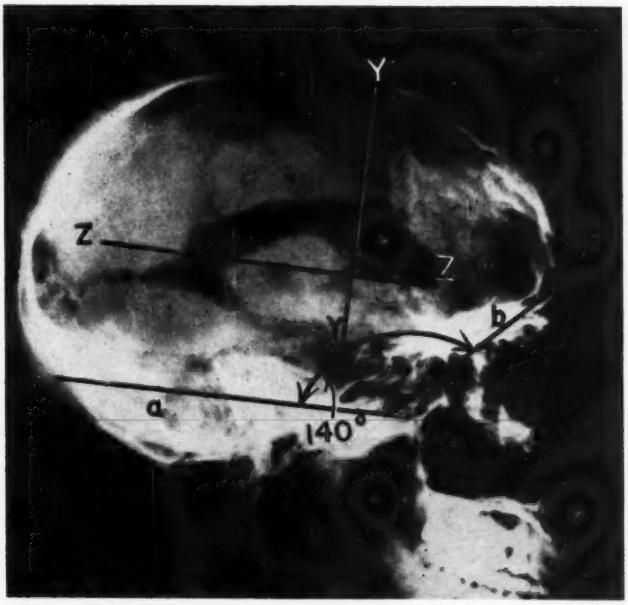


FIG. 2. Lateral view of a pneumoencephalogram showing the positions of the Y and Z axes passing through the anterior commissure parallel to line a, drawn from the internal occipital protuberance to the lowest point of the temporal fossa. The Y axis passes through the anterior commissure perpendicular to the Z axis, and the X axis (not shown) passes through the anterior commissure perpendicular to both the Y and Z axes. Rotation of both the Y and Z axes about the X axis approximately 15 degrees (in this view counterclockwise) would bring the Y axis into a vertical position and the Z axis into a horizontal position, thus facilitating the use of routine anteroposterior and lateral roentgenographic views.

respect to the Y axis ranged from 14 to 19 mm. and averaged 16 mm. The pallidum extended a mean distance of 9 mm. dorsal and 7 mm. ventral to the zero point.

Measurements taken with respect to the X axis indicated that the most medial border (ventromedial angle) of the globus pallidus was located at approximately the level of the anterior commissure. Distances from a mid-sagittal plane to the most medial border of the globus pallidus ranged from 6 to 12 mm. and averaged 9 mm. The most lateral border of the globus pallidus was located near its caudal limit. Distances from the mid-sagittal

plane to the most lateral border of the globus pallidus ranged from 23 to 28 mm. and averaged 26 mm.

Using plots similar to that shown in Fig. 3 it was possible to estimate the axis of orientation of the globus pallidus with respect to the mid-sagittal plane. The angle formed by the axis of the globus pallidus and the mid-sagittal plane measured in 11 normal brains varied from 30 to 40 degrees and averaged 34 degrees (Fig. 3, part B). Measurements of the globus pallidus made on 11 normal brains are recorded in Table 1.

The greatest variation was that of the rostrocaudal dimension of the globus pallidus, as measured along the Z axis. No apparent relationship existed between the dimensions of the globus pallidus and the weights of the brains. Measurements taken on 1 fresh brain were comparable to those taken on brains fixed in 20 per cent formalin. The globus pallidus of the brain fixed in 90 per cent alcohol was reduced in all dimensions, but the general configuration and orientation with respect to the established axes were unchanged.

By superimposing plots of the globus pallidus from the 11 normal brains, it was possible to determine the coordinates for what might be called the "geographic center" of the globus pallidus. The coordinates for this point were: 17.5 mm. lateral with respect to the X axis, 6 mm. posterior with respect to the Z axis and zero with respect to the Y axis. The 3 brains with pathology were omitted from the tabulation, but it is notable that the calculated "geographic center" of the globus pallidus obtained for these brains was the same as for the normal brains, although greater variation was seen in the pallidal configuration.

DISCUSSION

Recent experimental studies as well as clinical observations indicate that localized lesions of the globus pallidus may ameliorate and in some instances abolish dyskinesia and disorders of muscle tonus associated with certain specific extrapyramidal disorders. In the rhesus monkey violent choreoid activity resulting from localized destruction within the subthalamic nucleus was reduced or abolished contralaterally by lesions in the medial segment of the globus pallidus which destroyed approximately 10 per cent of the estimated total volume of the pallidum.[2] The percentile volume of destruction of the globus pallidus necessary to ameliorate tremor and rigidity in the human has not been determined, but inferentially it might be expected to be of the same order as in the rhesus monkey. Thus if the human globus pallidus has a volume of approximately 3000 c.mm.,[3] destruction of about 300 c.mm. of the globus pallidus would appear to be minimal. It should be noted that the physiologic effect of lesions in the medial segment of the globus pallidus is quantitatively greater than that resulting from lesions in the lateral pallidal segment because a greater number of pallidofugal fibers are interrupted.

According to Wycis and Spiegel[7] coordinate systems based upon intra-

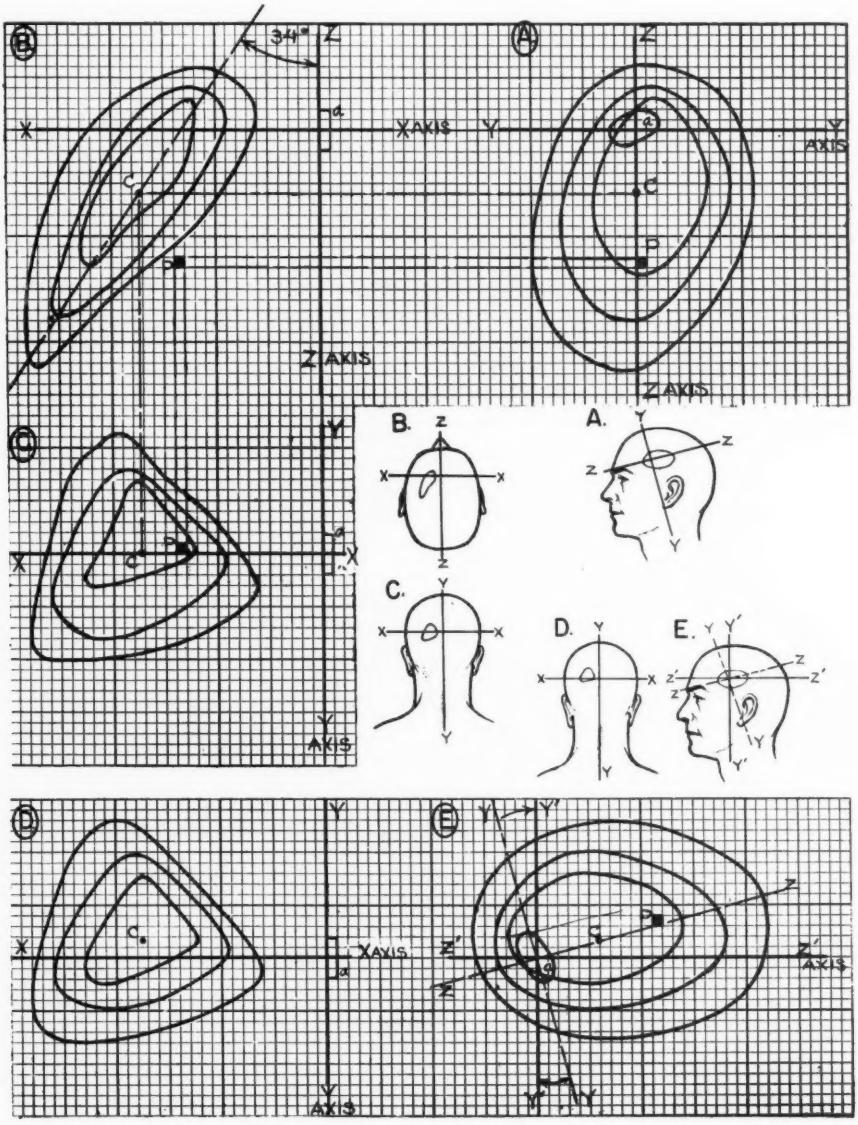


FIG. 3. Drawings A, B, and C represent lateral, dorsal and frontal projections of the globus pallidus with respect to the X, Y, and Z axes. Small letter a represents the position of the anterior commissure at its central part. The inner line delimits the dimensions of the minimum composite zone of the pallidum, a zone common to all the pallida in 15 brains. The middle line outlines the limits of the mean composite pallidal zone and the outer line defines the limits of the maximum composite pallidal zone. Point C repre-

TABLE 1 (part 1)

Dimensions of the globus pallidus with respect to the X, Y, and Z axes
X axis (dorsal projection)

Number in Series	Distance from a Mid-Sagittal Plane to the Medial Border of the Pallidum* (mm.)		Distance from a Mid-Sagittal Plane to the Lateral Border of the Pallidum† (mm.)		Axis of the Pallidum with Respect to a Mid-Sagittal Plane (Degrees)	
	R	L	R	L	R	L
1	8	9	27	25	39	30
2	7	7	24	23	37	33
3	9	12	27	28	32	30
4	7	7	24	24	33	33
5	9	9	26	26	35	34
6	10	10	27	27	33	33
7	9	9	26	25	35	33
8	10	9	28	28	40	36
9	9	6	24	28	36	40
10	8	7	24	24	32	34
11	10	12	26	25	34	33
Averages	8.7	8.8	25.7	25.7	35.1	33.0
Range	6–12		23–28		30–40	
Mean (Both Sides)	9±1		26±1		34±1	

* Minimum distances.
† Maximum distances.

cerebral structures offer the most constant and reliable reference data. The anterior commissure would appear to be the most satisfactory reference structure for stereotaxic approaches to the globus pallidus. Data regarding the spatial relationships between the anterior commissure and the globus pallidus have been presented. Although the number of brains measured in this study was relatively small, certain significant information concerning parameters resulted. The so-called "geographic center" and the minimum, mean, and maximum composite dimensions of the globus pallidus were delimited. It is noteworthy that in the brains measured these findings were not invalidated by pathological processes, including arteriosclerotic pallidal lesions.

---

sents the so-called "geographic center" of the globus pallidus. Point P, which lies within the minimum composite zone of the globus pallidus in the lateral and frontal projections, falls outside of the pallidum in the dorsal projection. This illustrates the importance of projecting the position of electrode placements from the anteroposterior and lateral views to the dorsal projection.

Drawings D and E represent frontal and lateral projections of the globus pallidus following rotation of the Y and Z axes approximately 15 degrees (in this case clockwise) about the X axis. This rotation orients the Y′ axis vertically and the Z′ axis horizontally. No change in the configuration of the pallidum results from this rotation in the lateral projection, and the change in the configuration of the frontal projection is small. The "geographic center" of the globus pallidus is shifted dorsally about 2 mm. by the rotation. The rotated Y and Z axes are designated Y′ and Z′.

TABLE 1 (part 2)

Y axis (*frontal projection*)

Number in Series	Dorsal Extent of the Globus Pallidus Above the Zero Point* (mm.)		Ventral Extent of the Globus Pallidus Below the Zero Point* (mm.)		Over-all Height of the Globus Pallidus* (mm.)	
	R	L	R	L	R	L
1	8	8	8	9	16	17
2	10	10	7	4	17	14
3	7	8	8	8	15	16
4	10	9	7	7	17	16
5	8	8	8	8	16	16
6	8	9	7	7	15	16
7	11	11	4	5	15	16
8	10	11	7	6	17	17
9	8	7	7	8	15	15
10	9	8	6	7	15	15
11	9	7	9	9	18	16
Averages	8.9	8.7	7.1	7.1	16.0	15.8
Range	7–11		4–10		15–19	
Mean (Both Sides)	9±1		7±1		16±1	

* Maximum distances.

TABLE 1 (part 3)

Z axis (*lateral projection*)

Number in Series	Extent of the Globus Pallidus Rostral to the Ant. Commissure* (mm.)		Extent of the Globus Pallidus Caudal to the Ant. Commissure* (mm.)		Over-all Length of the Globus Pallidus* (mm.)	
	R	L	R	L	R	L
1	5	4	16	20	21	24
2	4	4	15	20	19	24
3	4	3	20	20	24	23
4	5	3	15	21	20	24
5	3	6	18	19	21	25
6	5	5	19	21	24	26
7	3	3	23	19	26	22
8	3	2	14	17	17	19
9	6	4	17	15	23	19
10	5	5	17	19	22	24
11	5	4	17	17	22	21
Averages	4.4	3.9	17.1	18.9	22.6	22.8
Range	3–6		14–23		19–26	
Mean (Both Sides)	4±1		18±1		22±1	

* Maximum distances.

Assuming that the accuracy of the stereotaxic method is dependent upon both the coordinate system used and the volume of the "target" structure, all electrode placements made within the minimum composite dimensions of the globus pallidus with respect to the "geographic center" should lie within the globus pallidus. The minimum dimensions thus represent a pallidal zone common to all 15 brains measured. Similarly, electrode placements within the mean composite dimensions of the globus pallidus would lie within the globus pallidus in approximately 50 per cent of these cases, while electrode placements within the maximum composite pallidal dimensions, but outside of the mean composite zone, would lie within the globus pallidus in only about 10 per cent of these cases. No electrode placements outside of the maximum composite pallidal zone would fall within the globus pallidus.

Thus the problem resolves itself into placing the electrode within the dimensions of the minimum composite zone of the globus pallidus with the aid of roentgenographic techniques. To do this ideally three pneumoencephalographic views would be required: 1) a routine lateral, 2) an anteroposterior view taken perpendicular to the Z axis and 3) a dorsal view taken perpendicular to the Y axis. Since the third view cannot be obtained and in the second view the region of the anterior commissure would be obscured by bony structures, a more practical plan is necessary. On any good lateral view of a pneumoencephalogram the Y and Z axes can be identified and the position of the X axis determined. In any anteroposterior view the mid-line constitutes the Y axis. Thus, if an electrode placement in the globus pallidus is attempted, its position with respect to the X, Y, and Z axes can be determined from the lateral views and the distance of the electrode tip from the Y axis can be measured from the anteroposterior view. If such an electrode was located within the minimum composite pallidal zone, this would seem to be all that is required. However, a study of Fig. 3 shows that it is possible to place an electrode tip so that it would appear to be within the minimum composite pallidal zone in both the lateral and anteroposterior views and actually be situated outside of the globus pallidus because of the obliquity of the pallidum with respect to the mid-sagittal plane. In other words, without three views a "blind spot" exists which could be the source of considerable error. This difficulty can be circumvented by the use of the graphs shown in Fig. 3. If the position of the tip of the electrode in the lateral and anteroposterior views are plotted and then projected onto the dorsal view, the precise location of the tip of the electrode can be determined. If the position of the the electrode tip falls within the minimum composite pallidal zone in all views, the resulting placement lies within the globus pallidus.

In order to utilize the routine anteroposterior roentgenographic view it is necessary to change the position of the Y and Z axes by rotation with respect to the X axis, so that the Y axis becomes vertical, as shown in Fig. 3. This can be accomplished by rotating the Y and Z axes approximately 15 degrees (Fig. 3). Although no alteration in the configuration of the lateral projection results, slight changes are evident in the anteroposterior pro-

jection. The most significant change to occur is the displacement of the "geographic center" of the pallidum dorsally about 2 mm.

The method of determining the spatial relationships between the anterior commissure and the globus pallidus used here, of course, could be utilized for other intracerebral structures. Its use in basic animal investigations* has not been explored, but it might result in more economical use of time, animals and material.

The development of stereotaxic coordinate systems with respect to intracerebral structures identifiable by various roentgenographic techniques may result in simpler, more usable instruments than those now available.

SUMMARY AND CONCLUSIONS

A study of the spatial relationships of the globus pallidus with respect to the anterior commissure has been made using 15 human brains. A method for graphically representing the dimensions, spatial configuration and orientation of the globus pallidus with respect to the anterior commissure has been devised. This method can be used in a variety of ways to demonstrate other structural relationships within the brain. Data concerning variations in the dimensions of the globus pallidus determined by this method have been presented. A practical method† for utilizing this information in conjunction with pneumoencephalographic data to make electrode placements within the globus pallidus has been offered.

The following conclusions were drawn:

1. The "geographic center" of the globus pallidus, determined by superimposing plots of the structure in each of three different projections, was found to be: 6 mm. caudal to the anterior commissure, 17.5 mm. lateral to it, and at the same height (all measurements with respect to primary established axes).

2. A minimum composite pallidal zone, based upon pallidal dimensions common to all 15 brains in three projections, has been delimited as a zone within which all electrode placements would lie within the globus pallidus.

3. Electrode placements within the mean composite pallidal zone would lie within the globus pallidus in approximately 50 per cent of these cases, while electrode placements outside of the mean composite zone, but within the maximum composite zone of the pallidum, would fall within the globus pallidus in only about 10 per cent of these cases.

4. These data suggest that the accuracy and ease of electrode placement in the globus pallidus would be facilitated by information concerning the

---

* Since this report was written, this method has been used by one of us (E.M.H.) to plot the relationships between the caudate nucleus and the junction of the ansate and suprasylvian gyri in the cat for stereotaxic investigations.

† The accuracy of these coordinates has been confirmed stereotaxically in 2 fresh cadavers (personal communication from Drs. H. H. Rossi and J. B. Campbell). Data derived from Fig. 3 were helpful in determining the limits of the globus pallidus and the localization of an electrode within this structure in a clinical study done at the Neurological Institute. The location of a pallidal lesion in one patient of this series who subsequently died was verified at autopsy.

spatial relationships between the anterior commissure and the globus pallidus and that visualization of the anterior commissure by pneumoencephalographic means should become an integral part of the procedure.

5. Because of the oblique orientation of the globus pallidus with respect to the mid-sagittal plane, the position of placements determined from the lateral and anteroposterior views should be projected onto a dorsal view to avoid error.

6. The minimum composite pallidal zone appears to be the locus within the pallidum where lesions could be inflicted with the greatest constancy and the least risk.

The authors acknowledge with sincere thanks the cooperation of Dr. Abner Wolf of the Division of Neuropathology.

REFERENCES

1. Carpenter, M. B., and Whittier, J. R. Study of methods for producing experimental lesions of the central nervous system with special reference to stereotaxic technique. *J. comp. Neurol.*, 1952, *97:* 73–131.
2. Carpenter, M. B., Whittier, J. R., and Mettler, F. A. Analysis of choreoid hyperkinesia in the rhesus monkey. Surgical and pharmacological analysis of hyperkinesia resulting from lesions in the subthalamic nucleus of Luys. *J. Comp. Neurol.*, 1950, *92:* 293–331.
3. Harman, P. J., and Carpenter, M. B. Volumetric comparisons of the basal ganglia of various primates including man. *J. comp. Neurol.*, 1950, *93:* 125–137.
4. Horsley, V., and Clarke, R. H. The structure and functions of the cerebellum examined by a new method. *Brain*, 1908, *31:* 45–124.
5. Narabayashi, H. Okuma, T., and Shikiba, S. Procaine oil blocking of the globus pallidus. *Arch. Neurol. Psychiat., Chicago*, 1956, *75:* 36–48.
6. Spiegel, E. A., Wycis, H. T., Marks, M., and Lee, A. J. Stereotaxic apparatus for operations on the human brain. *Science*, 1947, *106:* 349–350.
7. Wycis, H. T., and Spiegel, E. A. Thalamotomy and mesencephalothalamotomy: neurosurgical aspects (including treatment of pain). *N. Y. St. J. Med.*, 1949, *49*, pt. 2: 2275–2277.

LAMINAGRAPHY WITH CEREBRAL PNEUMOGRAPHY*

CAPTAIN R. A. MANFREDI (MC), U.S.A.F.,† AND MAJOR F. KRUSE, JR. (MC), U.S.A.F.‡

(Received for publication October 25, 1956)

NUMEROUS procedures have been developed for the diagnosis and localization of intracranial pathology. Pneumography, arteriography, electroencephalography and radioactive isotope studies[2] have all been used with some measure of success. Cerebral arteriography has proved its greatest usefulness in the localization of vascular abnormalities. However, this procedure has not been uniformly successful in the diagnosis and localization of space-occupying lesions, and the incidence of complications does not permit its routine employment as a diagnostic procedure for such lesions.[4] Electroencephalography, while reliable in experienced hands, is not consistently accurate in the diagnosis and localization of intracranial neoplasms, particularly lesions not involving the cerebral convexities.[1] Cerebral pneumography has remained the single most important aid in the diagnosis and localization of tumefactions.

The procedure of pneumoencephalography has been modified variously to obtain better filling and visualization of the ventricular system.[5,7] These methods have all been concerned with variations in the technique of instillation of air and in the positioning of the head. The midline anatomy of the ventricular system has always been difficult to visualize with the usual techniques. The problem of obscuration and confusion by overlapping shadows of air in the lateral and midline ventricles, the cisterns and subarachnoid spaces and the overlapping bony shadows, persists. A satisfactory procedure that can overcome these difficulties, and one that has received little attention, is the addition of laminagraphy to the routine pneumograms.

The use of laminagraphy in neuroradiology has generally been limited to the visualization of the bony structures at the base of the skull. The use of this procedure in conjunction with air studies has been neglected and has not received full recognition of its potentialities. Pneumography is well suited to the laminagraphic studies. When air is placed in the ventricles, a system of contrasting densities results in an ovoid body surrounded by bone. With laminagraphy, even small quantities of air in any region of the ventricles can be identified accurately by the level of the fulcrum of transection. It results in an unusual clarity of image and any deformities become obvious. Laminagraphic studies with pneumography have appeared sporadically in the literature.[3,6] These reports have emphasized the value of this technique in

* Presented as part of a Scientific Exhibit at the annual convention of the American Medical Association, Chicago, Illinois, June 11–15, 1956.

† Present address: 912 South Wood Street, Chicago 12, Illinois.

‡ 3275th USAF Hospital, Parks Air Force Base, California.

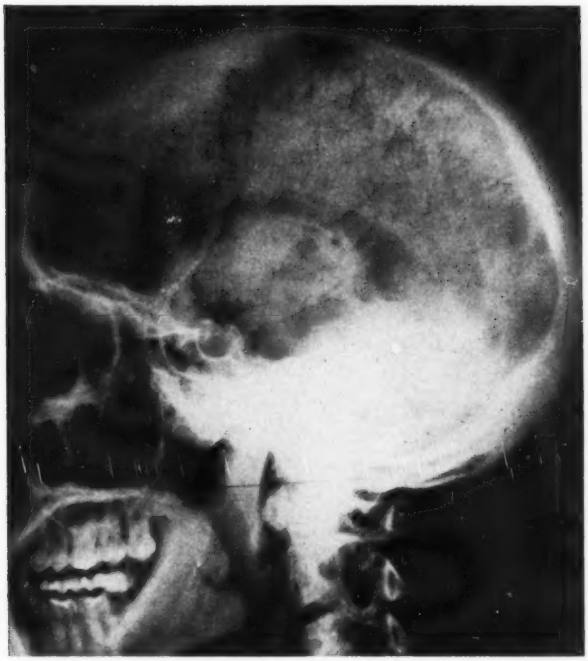


FIG. 1. *Case 1.* Plain lateral view of normal pneumoencephalogram showing air in the lateral ventricles.

the visualization of the midline structures: the 3rd ventricle, the aqueduct of Sylvius and the 4th ventricle. We have found this technique to be equally valuable to demonstrate deformities and displacements of various regions of the lateral ventricles not demonstrable by the routine and stereoscopic views. By using this technique, formerly unsuccessful pneumograms are now satisfactory and visualization of the ventricular air shadows at any desired site becomes adequate.

This technique entails no added risk or discomfort to the patient. No more than 40 cc. of air are necessary to obtain adequate visualization of the ventricular system. The additional equipment is available as part of the standard equipment of most departments of radiology. It is a relatively simple technique and requires only an additional few minutes of time to provide an exceptional visualization of the ventricular system and a greater clarity of image at the desired site.

TECHNIQUE

This method was used in ventriculography and pneumoencephalography. The technique of the latter procedure is that described by Lindgren.[5] By this method, not more than 40 cc. of air are necessary to adequately fill the ventricles. After the routine and stereoscopic radiographs are taken, the patient is placed horizontally

FIG. 2. *Case 1.* Midline laminagram taken at same air study of Fig. 1, clearly demonstrating the normal midline anatomy.

on the table in either the right or left lateral position. The midline plane is then measured from the table to the plane that transects the nasion and inion. The tube is placed 30 cm. above the fulcrum and travels superiorly and inferiorly a distance of 18 in. in 2 sec. at 60 to 70 kV. and 110 mA. The patient is then placed supine and another laminagraph is taken, directed over the sella turcica, with the tube travelling only 12 in. in the craniocaudad direction. These films are reviewed. If there is a shift or deformity of the ventricular system, additional lateral laminagraphs are taken of the involved side, which is placed uppermost. When this study is completed, one not only has a complete visualization of the entire ventricular system, but also an accurate evaluation of any deformity and displacement.

CASE REPORTS

The following cases are illustrative of the advantages of this procedure. It must be realized that laminagraphs lose considerable detail in photographic reproduction.

Case 1. Figs. 1 and 2 show pneumoencephalogram and laminagram taken at the same air study. In Fig. 2, the normal appearance of the foramen of Monro, the boundaries of the 3rd and 4th ventricle, the aqueduct of Sylvius, the interpeduncular cistern, the basilar artery, the pons and the outline of the cerebellum are clearly

FIG. 3. *Case 2.* Anteroposterior pneumoencephalogram showing a 4-mm. shift of the lateral ventricles to the left with no significant distortion of contour.

FIG. 4. *Case 2.* Plain lateral pneumogram showing confusing air shadow in region of 3rd ventricle.

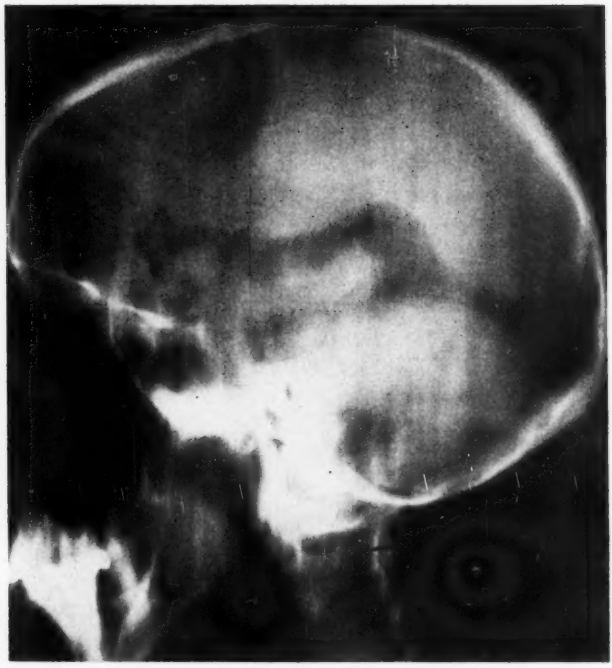


FIG. 5. *Case 2.* Lateral laminagram taken 3 cm. from the midline clearly demonstrates downward displacement of anterior portion of left lateral ventricle.

demonstrable. These two figures demonstrate well the advantages of laminagraphy. While the ventricles are adequately filled in Fig. 1, the overlapping air shadows and bony densities do not permit any visualization of the midline structures. With laminagraphy, the very small quantities of air in the midline ventricles and in the subarachnoid spaces are very clearly outlined and an excellent visualization of the surrounding anatomy is obtained.

Case 2. Figs. 3, 4, and 5 show the advantages of laminagraphy in the diagnosis of space-occupying lesions. This 38-year-old patient had a long history of hypertension and a 4-month onset of Jacksonian seizures. The spinal fluid pressure was normal and the protein content was within normal limits. There was a lack of response to antihypertensive therapy and pneumoencephalography was done. In the anteroposterior view (Fig. 3) there is a 4-mm. shift of the ventricular system to the right. The contour of the lateral ventricles does not appear unusually abnormal. In the lateral view (Fig. 4), one lateral ventricle is well outlined and appears normal. There is a confusing shadow in the region of the third ventricle which could not be well localized by stereoscopy. Laminagrams were then taken at 1-cm. levels through the abnormal side, and, at a distance of 3 cm. from the midline, a downward displacement of the left lateral ventricle was clearly seen (Fig. 5). At surgery, the deformity was found to be caused by a large meningioma. The excellent localization of the ventricular deformity by laminagrams avoided the necessity of additional procedures, such as arteriography.

FIG. 6. *Case 3.* Anteroposterior ventriculogram showing widely separated suture with a shift of both lateral ventricles, the left with the frontal horns shifted further than the 3rd ventricle, suggesting a right frontal lobe lesion.

Case. 3. The next patient was a 5-year-old child who had papilledema and bilateral 6th nerve palsy which developed over a 3-week period following measles. There were no localizing neurological signs. Ventriculography was done, and the anteroposterior view (Fig. 6) revealed a marked shift of the ventricular system to the left with the right lateral ventricle displaced further than the 3rd ventricle, suggesting a right frontal lobe lesion. In Fig. 7 the plain lateral view shows both frontal horns of the lateral ventricles with no shift in the lateral plane. The remainder of the lateral ventricle is not seen. By means of laminagraphy, at 3 cm. from the midline, the remainder of the body and occipital horn of the lateral ventricle are seen (Fig. 8), which are markedly elevated and outline a large lesion in the occipitotemporal area which proved to be an abscess at surgery. Without laminagraphic studies, a right frontal lobe lesion would have been suspected, and such a surgical exposure would have presented numerous complications without resolving the underlying pathology.

As demonstrated by the above cases, our experience with this technique has proved valuable. There is no risk involved in the procedure not already present in an air contrast study alone, and for the additional detailed information that can be obtained it appears well worth the time that is required for this technique.

CONCLUSIONS

1. Laminagrams in conjunction with air studies can be an important addition to the standard methods of investigation.

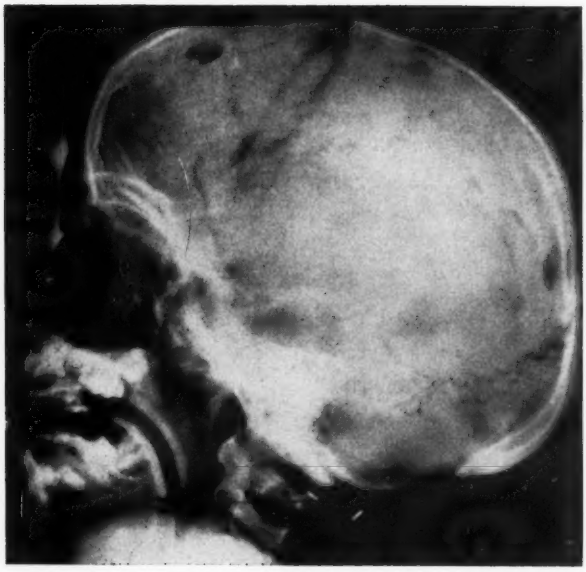


FIG. 7. *Case 3.* Lateral ventriculogram showing small amounts of air in the frontal horns without significant displacement in lateral plane.

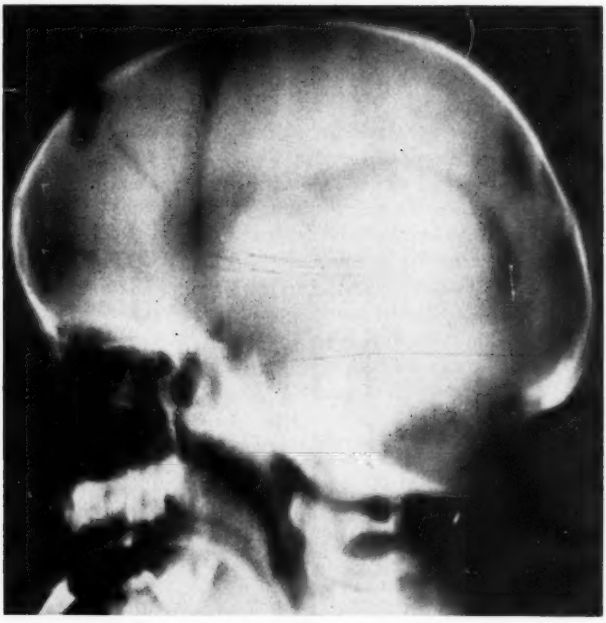


FIG. 8. *Case 3.* Lateral laminagram taken at 3 cm. to the right of the midline, showing a normally placed frontal horn and marked elevation of the body and occipital portion of the lateral ventricle outlining a lesion in the temporo-occipital area.

2. This technique is of particular value when pneumoencephalography is performed with small amounts of air.

3. A high incidence of visualization of the desired area with greater clarity of image and more detailed information can be obtained with laminagraphy.

REFERENCES

1. Blum, R. H. A note on the reliability of electroencephalographic judgments. *Neurology*, 1954, *4:* 143–146.
2. Davis, L., and Craigmile, T. Results of radioactive isotope encephalography in patients with verified intracranial tumors. *J. Neurosurg.*, 1954, *11:* 262–267.
3. Epstein, B. S., and Davidoff, L. M. The use of laminagraphy with encephalography in the diagnosis of midline and subtentorial brain tumors. *Amer. J. Roentgenol.*, 1946, *55:* 675–688.
4. Kaplan, A. D., and Walker, A. E. Complications of cerebral angiography. *Neurology*, 1954, *4:* 643–656.
5. Lindgren, E. Some aspects on the technique of encephalography. *Acta radiol., Stockh.*, 1949, *31:* 161–177.
6. Poppel, M. H., Jacobson, H. G., and Dewing, S. B. Value of the lateral mid-line tomogram in encephalography and ventriculography. *Radiology*, 1953, *60:* 236–240.
7. Shapiro, R., and Robinson, F. Controlled pneumoencephalography. A consideration of head position and gas-fluid replacement. *J. Neurosurg.*, 1954, *11:* 122–127.

POLAROGRAPHIC STUDY OF CORTICAL BLOOD FLOW IN MAN*

JOHN S. MEYER, M.D., AND JOHN HUNTER, M.D.†

Neurological Unit, Boston City Hospital, and Department of Neurology, Harvard Medical School, Boston, Massachusetts

(Received for publication November 14, 1956)

KNOWLEDGE of human cerebral hemodynamics in normal and diseased states has been advanced considerably by the application of the nitrous-oxide technique for measuring cerebral blood flow and metabolism.[4] This method provides an average measurement for the entire brain; the extent of localized circulatory and metabolic changes cannot be assessed. Moreover, the technique does not permit continuous recording of rapid changes in circulation or in metabolism.

In a search for a method whereby rapid adjustments of the cerebral circulation might be analyzed, the polarographic measurement of cerebral oxygen tension proved to be of value in the study of localized changes in blood flow and metabolism in such conditions as experimental cerebral concussion, contusion, laceration, brain-stem injury, ischemia, infarction and respiratory anoxia.[2,3,5–10] The polarographic method was also found to be of value in assessing local cerebral blood flow and metabolism in states of hypothermia, both in animals and in man.[11]

The historical background of the polarographic method and a critical assessment of its validity as an index of local blood flow and metabolism have been discussed in earlier communications.[7,10] The technique was first applied to physiological studies by Davies and Brink[1] in 1942 although it had been in use for many years in physical chemistry. The principle of electropolarography is that the oxygen tension of an unknown solution may be measured by recording the current flow between a platinum electrode acting as a cathode and a second nonpolarizable electrode. The local cerebral oxygen tension appears to depend on three factors: local blood flow, local metabolism of oxygen and the concentration of oxygen in the general circulation. If local tissue metabolism and the concentration in the general circulation remain constant, changes in local oxygen tension reflect alterations in local blood flow. It has been shown experimentally that cerebral metabolism and the concentration of oxygen in the general circulation remain remarkably constant under light anesthesia.[7]

* These studies were aided by a contract between the Office of Naval Research Department of the Navy, and Harvard University, NR 115-401, and a grant from the National Institute of Neurological Diseases and Blindness, B-1044, 1956–1957.

Opinions expressed are those of the authors and do not necessarily represent those of the Department of the Navy.

† With the technical assistance of Robert C. Struzziero.

We wish to report observations in man relating to cortical blood flow and oxygen metabolism determined by the polarographic method during the course of neurosurgical procedures. The method has been used in order to provide information relating to possible changes in blood flow and metabolism in cerebral contusion, cerebral laceration and cerebral compression from subdural hematomata. This information was correlated with the electroencephalogram as an indication of neuronal activity and with measurements of blood flow as indicated by temperature electrodes.[7] In addition, observations were made on patients with cerebrovascular disease in whom subdural hematoma was suspected and on patients with vascular malformations requiring occlusion of the carotid artery in the neck. For purposes of comparison, observations have been made in a case of juvenile dementia without signs of cerebrovascular disease.

MATERIALS AND METHODS

Thirteen cases were studied in this series; they are listed according to diagnosis in Table 1. In all instances the diagnosis was confirmed either by arteriogram, operation or necropsy. Polarographic measurements in 7 of these cases are illustrated and clinical summaries are included. The clinical and polarographic data obtained in Case 10 have been published in detail in another communication.[11] Measurements of cortical oxygen tension were made during the course of neurosurgical treatment, and in other cases the cortical oxygen tension was recorded after exploratory burr-holes had been made for diagnostic purposes (Cases 6, 7, 8, 13). These measurements were not found to be harmful and in some instances they were recorded under local anesthesia (Fig. 2). In the other cases, light anesthesia was maintained with endotracheal nitrous oxide and oxygen, occasionally supplemented with ether. Continuous charts of blood pressure were made by the anesthetist. The effects of changes in the oxygen content of the inspired air on the cortical oxygen tension were recorded while changes in the composition of the gas mixture were made by the anesthetist. In all cases except 2, electroencephalographic records were made during or a few hours prior to neurosurgery.

The oxygen tension of several small areas of brain was recorded concurrently with other parameters by modifications of the polarographic method described in earlier studies.[7,10,11] The anode (silver-silver chloride) is a silver wire insulated with Teflon, except for its last 1 cm., which is freshly chlorided prior to use. At operation the anode is inserted through a stab-wound situated on the edge of the craniotomy flap or the site of the burr-hole. The cathodes, or active electrodes, are several in number. Each is made from a 1-cm. length of platinum wire, 0.02 in. in diameter, soldered to an 8-cm. length of silver wire. The composite wire is insulated with Teflon except for the exposed tip of the platinum. The exposed portion is 3 mm. in length, tapered to a point and polished. The silver shaft of the electrode, being malleable, aids in placing the electrode in any desired area of cortex. The anode and several cathodes are soldered onto the adjustable rods of a

modified cortical electrode holder* which is autoclaved before each operation (Fig. 3). By means of this unit electrocorticograms can be concurrently recorded with measurements of cortical oxygen tension.

The principle of the polarographic method requires that a potential of 0.6 volt be supplied to the anode by a potentiometer.[10] The platinum electrode is connected to the positive pole of the potentiometer through a resistance of 1.2 million ohms. On either side of this resistance leads are taken to the input of a vacuum-tube millivoltmeter† which then functions as a microammeter. In some cases we have made concurrent polarographic records with the electroencephalograph and other parameters on an Offner 8-channel Type D3 polygraph (Figs. 2 and 7). In these records the output from the polarograph was wired directly to the Type 9138 Input Coupler of the inkwriter after calibration with a microammeter.

In some operations the polarograph electrodes were inserted through burr-holes. In these cases the cortical electrode holder was mounted on a tripod stand and fixed to the head by elastic bands clamped to the drapes. Before records were made the electrocautery machine was disconnected and movement in the vicinity of the operating field was reduced to a minimum as these were found to be a source of recording artifact. In some cases, changes in local blood flow were also measured by electrothermometry using a thermistor electrode placed on the cortex.[7]

Occlusion of the common or internal carotid artery was made by either a Selverstone or Blalock clamp, and in other instances by firm digital compression in the neck. The electroencephalogram was recorded either from the scalp or from the exposed cortex. In one instance (Case 2), an electropolarograph electrode and temperature electrode (thermistor) were implanted under the dura mater and daily records were made (Fig. 2). When concurrent measurements of cortical temperature were made, the temperature electrode consisted of sensitive microglass thermistor beads measuring .045 in. in diameter.[7] The thermistors were insulated with Teflon, and either implanted under the dura mater or mounted on one of the adjustable rods of the cortical electrode holder and placed on the cortex. Current (0.2 V.) for the thermistors was supplied by a 1.5 V. battery and potentiometer. Each electrode was wired into one arm of a Wheatstone bridge and any voltage drop was recorded with a vacuum-tube millivoltmeter or on one channel of the polygraph (Fig. 2I). Venous and cerebrospinal fluid pressures were recorded with strain gauges. Respirations were recorded with a pneumograph and strain-gauge assembly. Measurements of the oxygen saturation of the blood with an ear oximeter were made in only 1 case since it has been shown previously that with regular respirations, the cortical oxygen tension remains constant.[7]

---

* Grass Instrument Company, Quincy, Massachusetts.

† Model 17 CX, Millivac Instrument Corporation, Schenectady, New York.

TABLE 1

Case No. and Diagnosis	How Diagnosis Was Established	Age	Electroencephalogram	Operative Findings	Interpretation of Electroencephalogram, Polarogram and Temperature
1. Dementia and disseminated lupus erythematosus	Clinical findings, operation and biopsy	14	Alpha activity with rare bursts of triphasic slow waves	Normal cortex (brain biopsy)	Normal cortical blood flow and oxygen metabolism
2. Ruptured intracranial aneurysm, internal carotid artery-posterior communicating artery	Arteriogram. Blood in cerebrospinal fluid	42	Normal. Temporal slowing with carotid occlusion	Normal cortex	Active cortical oxygen metabolism. Poor cerebral collateral circulation
3. Chronic subdural hematoma	Operation	43	Left frontotemporoparietal reduction in amplitude	Large left-sided subdura ;hematoma with membranes; compressed brain	Impaired cortica lblood flow and oxygen metabolism in left frontotemporoparietal region
4. Cerebral contusion and subcortical hematoma	Operation	38	Left parieto-occipitotemporal reduction in amplitude, generalized slowing	Cerebral contusion and intracerebral hematoma, left temporal lobe	Impaired cortical blood flow and oxygen metabolism in left frontotemporal region
5. Embolic occlusion, middle cerebral artery	Operation	67	Right parietal reduction in amplitude	Softened yellow cortex in parietal region with pale veins and collapsed arteries	Edge of yellow zone showed sluggish blood flow; bordering zone showed good blood flow
6. Thrombotic occlusion, internal carotid artery	Necropsy	71	Not examined	Right frontoparietal yellowish softening with brain swelling	Diminished local blood flow and oxygen metabolism in softened area. Normal or increased blood flow on bordering area and intact hemisphere
7. Ruptured intracranial aneurysm, internal carotid artery	Arteriogram. Blood in cerebrospinal fluid	53	Normal	Right frontoparietal arteriovenous malformation with intracerebral blood clot	Low oxygen tension in center of hemorrhage. Normal or increased blood flow on border of lesion
8. Chronic subdural hematoma	Operation	82	Not examined	Moderate bilateral encapsulated subdural yellow fluid; compressed brain	Good blood flow, moderately reduced metabolism
9. Thrombotic occlusion, middle cerebral artery	Operation	65	Bitemporoparietal slowing	Swollen brain left temporoparietal region	Sluggish blood flow in swollen area
10. Arteriovenous malformation	Operation and arteriogram	57	Right central localized suppression with focal spiking	Right frontoparietal arteriovenous malformation with intracerebral blood clot	Low oxygen tension in center of hemorrhage. Normal or increased blood flow on border of lesion
11. Massive intracerebral hemorrhage	Operation and necropsy	65	Generalized slowing	Left temporal intracerebral blood clot and swelling	Low oxygen tension in hemorrhagic brain; no evidence of local blood flow. Impaired blood flow on border zone
12. Acute subdural hematoma	Operation	56	Right-sided reduction in amplitude	Recent subdural blood clot; compressed brain on right	Impaired blood flow and oxygen utilization, more marked on right side
13. Cerebral contusion	Operation	68	Right frontotemporal suppression. Generalized slow activity	Severely swollen, pulseless, contused brain in right frontotemporal region	No polarographic evidence of local blood flow in contused zone

GENERAL OBSERVATIONS

Measurements of availability of cortical oxygen in man are found to be similar in most respects to those reported previously for experimental animals.[2,3,5–11] The cortical oxygen tension is higher when the polarograph electrode is placed on an artery than when it is placed on a vein, while in the tissue space between artery and vein the readings are lower. In areas of old blood clot and necrotic brain caused by trauma or vascular disease the availability of oxygen was found to be low. In freshly infarcted brain the readings are usually low (Fig. 6) but on the border zone of the softened area, the readings are the same or higher than in undamaged regions of brain.

CHANGES IN COMPOSITION OF INSPIRED AIR

In normal cortex an increase in the oxygen composition of the inspired air is promptly indicated by a small rise in the readings recorded from the cortical polarograph electrodes (Fig. 1, and EPG 6, 7, 8 in Fig. 6). In areas of traumatic necrosis and infarction, or in areas of old hemorrhagic blood clot, the oxygen electrodes show no response to changes in the gaseous content of the inspired air. In bordering areas of ischemic or traumatized brain the electrodes record a recognizable but reduced response to changes in the inspired air (Figs. 4 and 5, and EPG 1 and 5 in Fig. 6).

VASCULAR OCCLUSION

The effects of manual occlusion of the common carotid artery upon the human brain with a normal cerebral blood supply are shown in Case 1.

Case 1. A 14-year-old girl was referred for diagnostic evaluation of juvenile dementia. A sister suffered from a milder degree of the same disorder. The electroencephalogram showed alpha activity with rare diffuse bursts of slow waves. Cortical biopsy showed normal cortex with thickened meninges.

Manual occlusion of the right common carotid artery at operation (Fig. 1) resulted in a transient fall in the cortical oxygen tension lasting 20 sec.; thereafter, there was a fluctuating return towards the steady state *before the vessel was released.* This phenomenon, termed "escape," has been studied in detail in experimental animals while observing the cortical blood flow with a microscope and measuring changes in cortical temperature.[3,6,7,9,10] The rise in oxygen tension prior to release is dependent on the cerebral collateral circulation.

An inadequate cerebral collateral circulation following rapid occlusion of the left internal carotid artery is illustrated in Case 2.

Case 2. A right-handed woman was admitted to hospital complaining of headache, stiff neck, deviation of the head and eyes to the left, right facial weakness, dysphasia and vomiting. Papilledema was present. The cerebrospinal fluid pressure exceeded 500 mm. of water and was grossly bloody. A left carotid arteriogram showed a 5-mm. saccular aneurysm at the junction of the internal carotid artery and the left posterior communicating artery. During arteriography there developed a brief episode of right hemiparesis and dysphasia, with complete recovery after 2 hours.

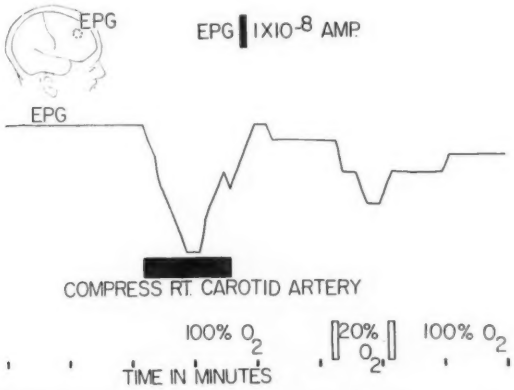


FIG. 1. *Case 1.* Electropolarographic (EPG) measurements of cortical oxygen tension in right frontal cortex of a 14-year-old girl with progressive dementia and without vascular disease. Right common carotid artery was occluded in neck by compression for 80 sec. Cortical oxygen tension fell 23% within 42 sec. and then began to rise prior to release. When vessel was released, cortical oxygen tension returned to steady state. Brief breathing of 20% oxygen after breathing 100% oxygen also produced a fall in cortical oxygen tension. Blood pressure throughout was 110/80. Calibration for polarograph is at top of record. Position of EPG electrode is shown on diagram of brain. Calibration of EPG in electrical units is at top of record. Time intervals of 1 min. are marked at bottom of record. Local anesthesia was supplemented with endotracheal nitrous oxide, ether and oxygen.

The records in Fig. 2 were made after a Selverstone clamp had been placed around the left internal carotid artery. At Fig. 2A the clamp was closed for 12 sec., causing a severe reduction in the cortical oxygen tension recorded by polarography through a burr-hole made in the left Sylvian region. The electroencephalogram showed slowing in this area also. Fig. 2B is a sample trace made 20 min. after the clamp was released. Fig. 2C was recorded as the clamp was again closed while the patient counted aloud. After 30 sec. the oxygen tension approached zero and high-amplitude slow waves appeared in the electroencephalogram recorded from the left hemisphere. The patient was unable to count further. After 80 sec. there was no evidence of any collateral circulation and the clamp was released (Fig. 2D). One minute after release of the internal carotid artery the polarogram showed a supernormal phase and the electroencephalogram improved. Fig. 2F shows records made as the clamp was partially closed by 2 turns ($8\frac{1}{2}$ turns closed the clamp completely). One min. later (Fig. 2G) the availability of cortical oxygen returned to the steady state and remained unchanged 4 min. later (Fig. 2I). Each day after operation records were made of the cortical oxygen and the cortical temperature from the same region while the clamp was closed progressively in small increments. On the 7th day (Fig. 2I) the clamp was closed completely without change in ability to count, respiration, electroencephalogram or cortical oxygen tension. The cortical temperature showed a small transient reduction as the clamp was being closed.

CEREBRAL TRAUMA

In patients with chronic subdural hematoma and compressed cortex, a minimal change in the cortical oxygen tension occurs when changes are made in the gaseous composition of the inspired air or when the common

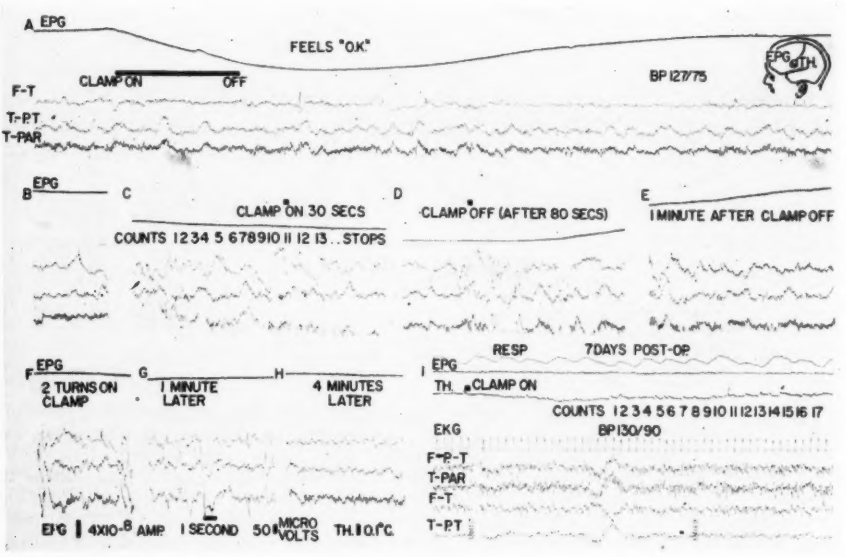


FIG. 2. *Case 2.* Polygraph of a woman, aged 42, suffering from a ruptured intracranial aneurysm of left internal carotid and posterior communicating arteries, made under local anesthesia. EEG, EKG, BP, availability of cortical oxygen (EPG) and cortical temperature (TH.) recorded from left cerebral hemisphere while left internal carotid artery was occluded with a Selverstone clamp. Position of burr-hole through which EPG and TH. electrodes were inserted is shown in diagram of brain. At A, the clamp was closed for 12 sec. A severe fall in availability of cortical oxygen from 16 to 6×10–8 Amp. resulted and EEG showed temporoparietal slowing. After release of clamp, availability of cortical oxygen returned to steady state. At B, clamp was again closed. The cortical EPG approaches zero without recovery, indicating absent collateral circulation. Thirty sec. later (C) slowing appeared in EEG and patient stopped counting. Clamp was released after 80 sec. (D). One min. after release of clamp (E), EPG recorded supernormal phase and EEG showed less slow activity. At F, the clamp was given 2 turns, resulting in transient reduction of availability of oxygen, then an increase within 1 min. (G) and finally returned to steady state (H). Clamp was gradually closed further each day. On 7th postoperative day clamp was completely closed (I). At this time there was neither reduction in availability of cortical oxygen, EEG change or impairment of speech. All calibrations shown at bottom left of record. F = Frontal, T = Temporal, P.T. = Posterior Temporal, Par = Parietal.

carotid artery is occluded. For example, Fig. 3 is a record made at operation for removal of a chronic subdural hematoma (Case 3).

Case 3. A 43-year-old male alcoholic was admitted from a prison hospital in a stuporous state. He had shown progressive confusion and drowsiness for the preceding month following a bout of delirium tremens. After admission to the Boston City Hospital his mental status varied between stupor and semistupor and he had a right spastic hemiparesis and papilledema. A large encapsulated subdural hematoma filled with fluid was removed at operation.

The electroencephalogram recorded from the compressed brain was of diminished amplitude (Fig. 3). A thermistor electrode placed on the cortex failed to show the normal degree of reduction in temperature when the carotid artery was occluded in the neck.

The record in Case 3 may be compared with that of Case 10, in which operation was performed for the removal of an arteriovenous malformation, which has been published previously.[11] In Case 10 there was a reduction in cortical temperature of over 1°C. in the Sylvian region when the common carotid artery was occluded temporarily.

In the patients with chronic subdural hematoma the cortex was compressed and the arteries were visibly reduced in size. In instances in which a hemisphere had been compressed, the records made with thermistor and

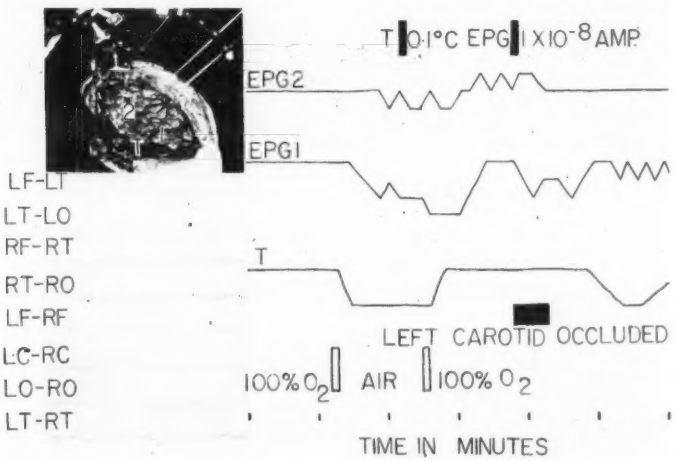


FIG. 3. *Case 3.* Records of cortical oxygen tension (EPG) and EEG of a 43-year-old man suffering from left-sided chronic subdural hematoma. At upper left is photograph made at operation showing compressed brain, part of the subdural clot and temperature and oxygen electrodes held in place by cortical electrode holder. EEG below photograph shows reduction in amplitude over left hemisphere. The response of cortical oxygen and temperature electrodes to breathing of air and carotid compression is reduced. Calibration of cortical temperature (T) and EPG is at top of record. Anesthesia was endotracheal oxygen and ether. Time intervals of 1 min. are marked at bottom of record.

polarograph electrodes appear to indicate that there is both a reduction in cortical blood flow and impaired neuronal activity. This interpretation is based on the facts that with carotid compression the thermistor record indicates a reduced blood flow, the electroencephalogram shows evidence of impaired neuronal function and the minimal polarographic changes may reflect either one or both of these factors. In our experience, in cases of acute subdural hematomata that are not complicated by contusion and laceration and in which the cortex is not massively compressed there is not the same degree of impaired neuronal metabolism.

Cerebral contusion and laceration with intracortical blood clot appear to cause a more localized disturbance of blood flow and metabolism, as shown by Case 4.

Case 4. A 38-year-old male suffered a fracture of the left temporal bone, a left temporofrontal contusion and intracerebral hematoma when he was struck on the head with a blunt instrument. He was semistuporous upon admission to hospital and mumbled unintelligibly. The tendon reflexes were increased on the right side, and the right plantar reflex was extensor. The electroencephalogram showed reduction of amplitude in the left frontal, temporal and parietal regions. A left temporal craniotomy revealed a left temporofrontal contusion and a fresh hematoma was evacuated from the temporal lobe.

Within the zone of necrotic brain and within the blood clot there was no observable change in the electrode readings when the carotid artery was occluded nor did the electrodes record any effect with change in the gaseous composition of the inspired air. The edge of the injured zone showed a diminished response when the oxygen content of the inspired air was increased and a slow gradient of fall when the carotid artery was occluded, with a failure of recovery when the vessel was released (Fig. 4, EPG 2). In areas further removed from the lesion the changes in cortical oxygen tension with these maneuvers approached the normal pattern (Fig. 4, EPG 1).

CEREBRAL THROMBOSIS AND EMBOLISM

Studies of the edge of an area of cortical infarction caused by embolism or thrombosis of a branch of the middle cerebral artery gave results comparable to those observed in cerebral contusion except that around the

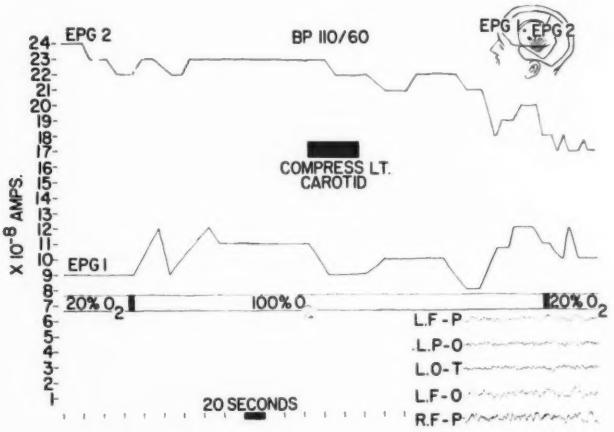


FIG. 4. *Case 4.* Record of cortical oxygen tension made in left cerebral cortex of a 38-year-old man suffering from left temporofrontal contusion and intracerebral hematoma (cross-hatched area in diagram). Record EPG 1 was made from left frontal region on edge of injured area and EPG 2 was a concurrent record from just inside contused region. The bordering zone (EPG 1) showed increase in cortical oxygen tension when oxygen was breathed and transient reduction when left common carotid artery was compressed. The injured area showed no EPG response to breathing of oxygen and delayed reduction in oxygen tension with impaired recovery when left common carotid artery was compressed briefly. EEG shows reduced amplitude from injured area with higher-amplitude slow waves surrounding lesion. EPG readings are at left of record; time marker indicates 20-sec. intervals. Anesthesia was endotracheal nitrous oxide, ether and oxygen.

infarct the area of impaired blood flow was more sharply delineated and the bordering zone showed a normal or increased blood flow, probably because of an active collateral circulation. Case 5, for example, shows the records made in a man who had sustained an embolic infarction in the Sylvian region and was operated upon to exclude the possibility of subdural hematoma.

Case 5. A 67-year-old hypertensive male suffered a myocardial infarction 2 weeks prior to admission to the Boston City Hospital. He suddenly lapsed into semistupor after falling and sustaining a laceration of the scalp. He showed a mild dysphasia, left homonymous hemianopia, a flaccid left hemiplegia, left hypesthesia, and hypalgesia from which he made an incomplete recovery.

In Fig. 5, the shaded area shown in the diagram of the brain was softened and yellow with pale veins and collapsed arteries. EPG 1 was placed on the edge of this area and EPG 2 was placed near an ascending frontal branch of the middle cerebral artery, which appeared normal. Manual occlusion of the right common carotid artery resulted in a more severe fall in EPG 1 than in EPG 2, with a prolonged phase of recovery. EPG 2 showed a prompt recovery with a supernormal phase which is believed to indicate increased blood flow in the ascending frontal branch of the middle cerebral artery, which was supplying the area in which the electrode was placed.

When cerebral infarction is caused by thrombotic occlusion of the internal carotid artery the area of infarction has a less well defined border than in those cases in which a smaller vessel is occluded, presumably because of a larger collateral circulation. Case 6 presents samples of records made in such a case.

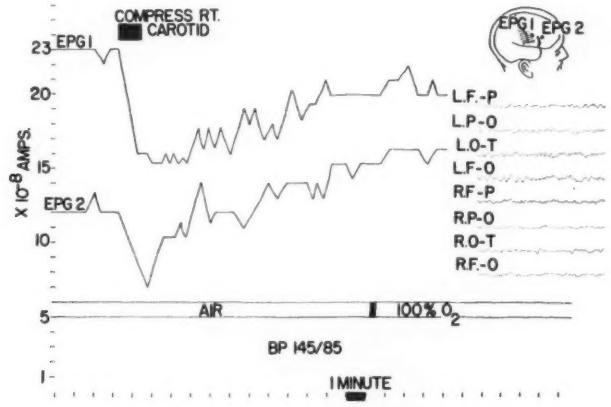


FIG. 5. *Case 5.* Measurements of cortical oxygen tension made during operation under local anesthesia to exclude subdural hematoma in a 67-year-old man who had suffered from embolic occlusion of right middle cerebral artery 3 days before operation. Shaded area indicates softened, yellow cortex with pale veins and collapsed arteries noted at operation, on the border of which EPG 1 was placed. EPG 2 was placed near an ascending frontal branch of middle cerebral artery, which showed a good flow. Compression of right common carotid artery caused a more prolonged reduction in oxygen tension recorded from EPG 1 than EPG 2. EPG readings are indicated at left of record. Time marker indicates intervals of 1 min. at bottom of record.

*Case 6.** A 71-year-old man proved at necropsy to have a thrombotic occlusion of the parasellar portion of the right internal carotid artery in the neck. He was known to have had both diabetes and hypertension. He entered the Boston City Hospital after being struck by a car, suffering severe fractures of the pelvis and the left tibia and fibula. During reduction of the fractures the blood pressure fell to shock levels and a left hemiparesis developed, with hemianesthesia and Cheyne-Stokes respiration. The pulse rate increased from 140 to 156 and a state of impending shock was apparent. The periods of apnea became prolonged and the mental status varied with the respiratory cycle. During apnea he became comatose but when respirations resumed he began to respond within 15 sec. During the periods of hyperpnea he was able to give a brief history of the accident and said that while crossing the street he felt dizzy and collapsed. After a transfusion of 1,000 cc. of blood the systolic blood pressure rose to 120 mm. of mercury, normal respirations were resumed and he was able to converse normally. Examination at this time showed paralysis of conjugate gaze to the left, severe spastic left hemiparesis, left hemihypesthesia and sensory inattention on the left to bilateral simultaneous stimulation. Both plantar reflexes were extensor. The blood pressure fell again temporarily and the patient lapsed into coma with Cheyne-Stokes respiration from which he never recovered. There was marked carotid sinus sensitivity on the right. At operation bilateral burr-holes were made to exclude the possibility of a subdural hematoma.

The polarographic records made at operation are shown in Fig. 6. In the softened area of cortex the response to reduction in the oxygen content of the inspired air is minimal (EPG 1 and 5), while electrodes placed in the cortex of the intact hemisphere showed a normal response (EPG 6, 7 and 8). In the border of the infarct the electrodes showed a variable response. Brief reduction in blood pressure caused by massage of the carotid sinus resulted in a marked but patchy reduction in the availability of cortical oxygen recorded by the polarograph electrodes in the right hemisphere, while those in the left hemisphere showed a uniformly rapid gradient of oxygen reduction. The greater reduction of oxygen tension recorded by EPG 4 placed near a small artery in the border of the softened zone is presumably because this vessel was providing a critical blood supply to the area.

ARTERIAL OCCLUSION WITH REDUCED CEREBRAL METABOLISM AND GOOD COLLATERAL CIRCULATION

Polygraphic records made during closure of the right common carotid artery in one stage under hypothermia are presented in Case 7.

Case 7. The patient was transferred from another hospital after suffering three episodes of subarachnoid hemorrhage from which a satisfactory recovery was made. There were no abnormal neurological signs other than a dilated pupil on the right side. A right-sided carotid arteriogram showed a large (1.5 cm.) saccular aneurysm of the parasellar portion of the internal carotid artery. At operation the right common carotid artery was occluded with a Selverstone clamp in the neck.

The records shown in Fig. 7 were made during the induction of hypothermia. There was a reduction of blood pressure from 128/86 at 35°C. to 90/70 at 33°C. The cerebrospinal fluid pressure was reduced from 150 to 125 mm. of water and the venous pressure also decreased. The electroencephalogram was not significantly altered (Fig. 7A, B). Positive-pressure breathing caused a rise in blood pressure and

* Dr. Edward L. Spatz kindly made this patient available for study.

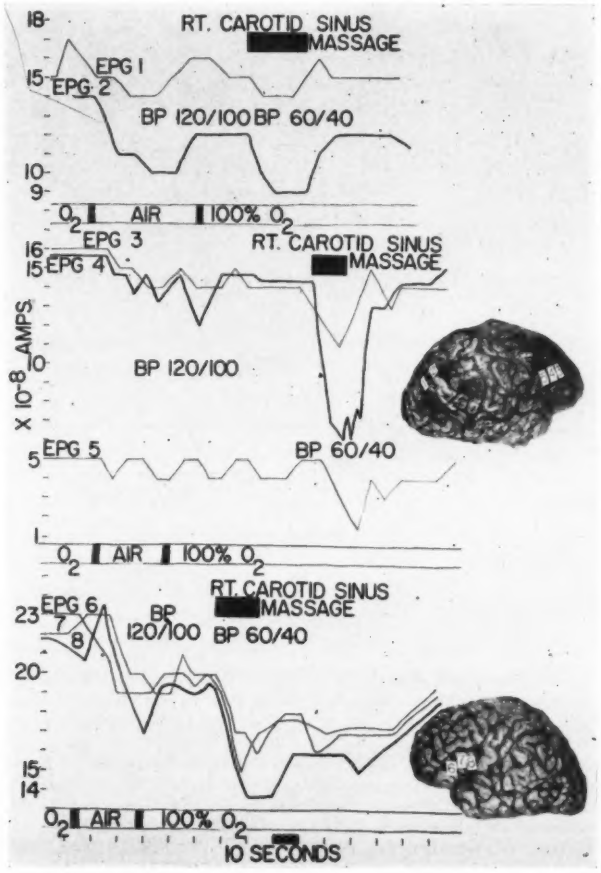


FIG. 6. *Case 6.* Records of cortical oxygen tension (EPG) from several areas of both cerebral hemispheres made at operation, under local anesthesia, on a 71-year-old man with thrombosis of right internal carotid artery. Blood pressure was reduced from 120/100 to 60/40 by massage of carotid sinus. Electrodes were inserted through multiple burr-holes made to exclude subdural hematoma. Positions of electrodes are shown on photographs of brain at necropsy. EPG 4 was placed near a small artery. Softening was present in distribution of right middle cerebral artery. In softened area response to reduction of oxygen content of inspired air is minimal (EPG 1, 5), and in cortex of intact hemisphere there is normal response. A fall in blood pressure results in reduction in cortical oxygen tension. EPG readings are higher in intact cortex than in softened area. EPG units are recorded to left of record; time marker indicates 10-sec. intervals.

in venous and cerebrospinal fluid pressures (Fig. 7C). Occlusion of the common carotid artery by a Selverstone clamp resulted in a rapid, transient rise in blood pressure from 120/90 to 140/90 (baroreceptor reflex). The cortical polarogram showed an increase in oxygen tension coincident with the rise in blood pressure. After 15 min. a small reduction of oxygen tension in the Sylvian area occurred without further decrease in the following hour. The electroencephalogram remained un-

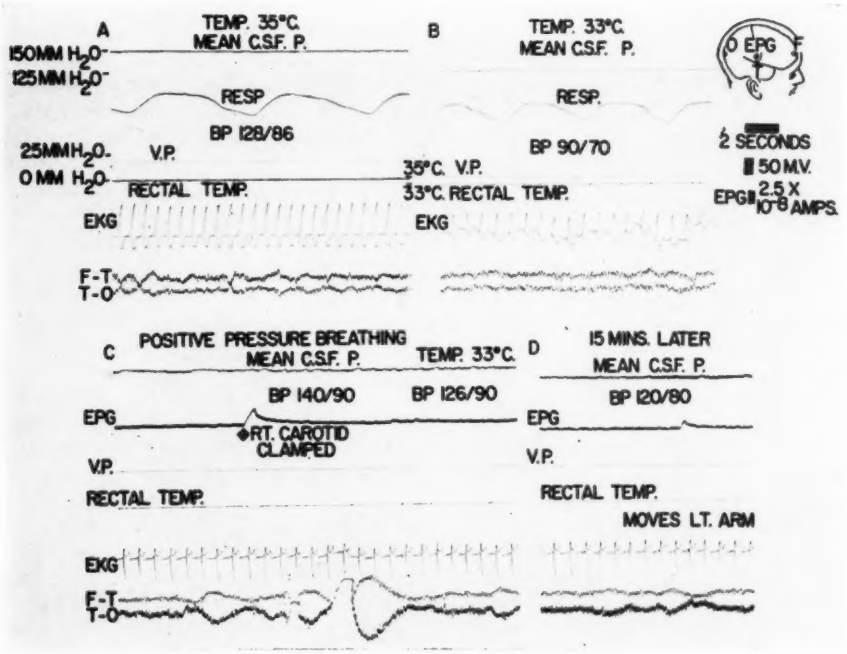


FIG. 7. *Case 7.* Polygraph made at operation on a 53-year-old woman in whom the right common carotid artery was clamped for ruptured aneurysm of right internal carotid artery, under hypothermia at 33°C. A is sample of continuous records of cerebrospinal fluid pressure, respiration, blood pressure, venous pressure, rectal temperature, EKG and EEG at 35°C. B is sample made at 33°C., showing reduction in venous CSF, and blood pressures. In record C a cortical polarograph (EPG) electrode was placed in right Sylvian region, and right common carotid artery was clamped. Blood pressure rose to 140/90. Positive pressure respirations were given by anesthetist, resulting in a rise in both CSF and venous pressures. There was no significant change in availability of cortical oxygen or in EEG; 15 min. later there was reduction of cortical oxygen tension from 20×10-8 Amp. to 17.5×10-8 Amp. Strength in right arm was normal at this time and subsequent recovery was excellent for all records. Calibration of CSF and venous pressures are to left of record A, and of thermistor in rectum to right of record A. Time, EEG and polarograph calibrations are right of record B. EPG and EEG positions are shown in diagram. Anesthesia was Demerol, Thorazine and endotracheal nitrous oxide and oxygen mixture.

changed and voluntary movement of the left arm and hand was normal. During observation for 8 months since operation there have not been any signs of neurological deficit.*

DISCUSSION

These observations made by placing a polarograph electrode on the human cerebral cortex, confirm previous observations made in experimental animals. The polarograph electrode appears to measure a value of tissue oxygen on a gradient between the blood vessel and neurone. This value is a varying quantity which is dependent on the amount of oxygen carried by

* This patient was operated upon by Dr. Walter Wegner; the other patients were operated upon by one of the authors (J.H.).

the arterial blood, the rate of local blood flow and the rate of utilization of oxygen by tissue. Under light anesthesia, or in the resting state, the cortical neuronal oxygen consumption remains at a remarkably stable level (Figs. 2 and 7). Changes in the oxygen composition of the inspired air cause parallel changes in the availability of tissue oxygen with a time lag of 5–30 sec. (Figs. 1, 3, 5, 6 and 7). The greater the degree of vascular damage, the longer the time interval. In areas of cortical necrosis, or old encapsulated blood clot, changes in the oxygen content of the blood are not reflected in the electrode readings since these areas have no blood supply. At the edge of an injured zone varying degrees of impaired blood flow are seen. Areas of severe brain compression also show a reduced cortical blood flow.

Occlusion of a main cerebral vessel causes a varying degree of local cortical hypoxia, the severity of which depends on the effectiveness of the collateral circulation and the rate of consumption of tissue oxygen. In the absence of cerebrovascular disease or severe compression of the brain, occlusion of the common carotid artery in the neck results in a transient cortical hypoxia in the distribution of the middle cerebral artery and compensation begins within 40 sec. (Fig. 1). In our experience, stenosis or thrombosis of an important collateral vessel, such as a partially thrombosed congenital saccular aneurysm of the posterior communicating artery (as in Case 2, Fig. 2), may result in poor compensation of the collateral circulation. In Case 2 there was no polarographic evidence of compensation after 80 sec. of occlusion of the internal carotid artery in the neck and clinical and electroencephalographic signs of localized cortical anoxia ensued. In such a patient reduction of the caliber of the internal carotid artery by one-quarter (as in Fig. 2F, G, H) was adequately compensated within 1 or 2 min. When the vessel was occluded in daily stages, sufficient increase in the collateral circulation had developed on the 7th day to permit total occlusion of the vessel without causing localized anoxia, disordered cerebral function or a change in the electroencephalogram (Fig. 2I). These observations confirm similar clinical observations made by others.[13]

Severe compression of the brain with visible collapse of the small cortical arteries (Fig. 3) associated with functional cortical paralysis and focal absence of electroencephalographic signs gravely decreases both neuronal oxygen supply and demand. Thus, when the common carotid artery is occluded in the neck there is no significant change in the measurements of cortical oxygen, presumably because demand for oxygen has been reduced to near zero. In Fig. 3 a small fall in cortical temperature, when the carotid artery was occluded, and the limited reduction in cortical oxygen tension induced by anoxic anoxia indicate that cortical blood flow was also critically reduced.

In cases of acute subdural hematoma with less marked cerebral compression we have found that utilization of neuronal oxygen is still present but recovery of the cortical oxygen tension following carotid occlusion may be delayed and incomplete 6 min. after the vessel is released.

In thrombotic or embolic occlusion of the middle cerebral artery and its branches there appears to be a circumscribed area of softened brain with absent or diminished circulation surrounded by a bordering zone in which the blood flow is normal or increased. A supernormal phase is frequently seen in areas that have been rendered severely anoxic by temporary occlusion of a cerebral vessel. This supernormal phase is not seen with brief occlusion of a vessel for 12 to 20 sec. but is marked when a vessel has been occluded for 80 sec. or longer (Fig. 2). The supernormal phase is also seen after temporary ischemia in experimental animals,[3,7,9,10] and appears to be caused by reactive hyperemia with an increased local circulation.

THE IMPORTANCE OF BLOOD PRESSURE IN MAINTAINING CEREBRAL BLOOD FLOW

It has been shown that electroencephalographic changes may be induced by reduction of the cardiac output in individuals with occlusion or stenosis of the basilar or carotid arteries.[12] The electroencephalographic changes, in general, are localized to the cerebral territory supplied by the diseased vessel. In experimental animals, lowering the blood pressure to 50 mm. of mercury in the presence of an occluded middle cerebral artery results in severe localized cerebral anoxia and eventually in infarction.[3,7,9,10] The observations recorded in Case 6 (Fig. 6) permit further analysis of this phenomenon. At necropsy an organized thrombus occluded the sigmoid portion of the right internal carotid artery above the foramen lacerum. All vessels of the circle of Willis showed marked atherosclerosis and there was patchy infarct necrosis in the distribution of the right middle cerebral artery. In Fig. 7, a marked diffuse decrease in the availability of cortical oxygen resulted from lowering of the blood pressure by massage of the carotid sinus. EPG 4 placed near a small artery in the bordering zone showed a reduction in availability of cortical oxygen to near zero levels. Had the local demand for oxygen not been reduced in a patchy manner by cerebral infarction it is probable that all the electrodes in the distribution of the right middle cerebral artery would have shown changes comparable to EPG 4, since infarction reduces tissue demand for oxygen.[2,3,9,12] A reversible, controlled reduction of consumption of neuronal oxygen may be produced by hypothermia,[11] and in this situation occlusion of the carotid artery in the neck may be carried out rapidly with a minimal and delayed reduction in availability of cortical oxygen and without functional impairment.

In other studies[5] it was concluded that cerebral contusion and laceration cause damage to cerebral blood vessels and result in cerebral hypoxia and acidity. Around the damaged brain a bordering zone of hyperemia was frequently demonstrable. In the present study the contused area illustrated in Fig. 3 was 3 days old. The pattern of response of the oxygen electrodes inside and at the border of the area of injury is similar in many respects to that of an area of infarction. In the damaged area there was evidence of de-

creased blood flow and utilization of oxygen with a transition zone between normal and abnormal brain.

RELATION OF CORTICAL ISCHEMIC ANOXIA TO CHANGES IN ELECTROENCEPHALOGRAM

Previous observations during vascular occlusion in experimental animals demonstrated a close correlation between reduction of cortical oxygen tension and the appearance of slow waves in the electroencephalogram.[6,7,10] In a study of a group of patients suffering from cerebrovascular disease, the rapidity of onset of electroencephalographic changes produced by lowering the blood pressure or by compression of the carotid artery in the neck was taken as strong evidence that localized cortical hypoxia was their cause.[12] In Fig. 2 concurrent records of the electroencephalogram and cortical oxygen tension during cerebral ischemia show a fairly close relation between the fall in cortical oxygen tension and the appearance of slow waves in the electroencephalogram. However, the electroencephalographic electrodes were widely spaced and were recording only approximately from the same area as the oxygen electrode.

DELAYED DEVELOPMENT OF CEREBRAL COLLATERAL CIRCULATION

The polygraph tracings illustrated in Fig. 2 are part of a large number of daily records made in Case 2. In the operating room it was found that if the internal carotid artery was reduced in caliber by one-half, the polarograph electrode recorded an incomplete compensation in the cortical blood flow. On the 2nd postoperative day the vessel was reduced in caliber by one-half without any evidence of defective collateral circulation as indicated by the thermistor and oxygen electrodes. By the 7th day the vessel was completely closed without producing any significant alteration in electroencephalogram, cortical oxygen tension or temperature. Presumably this indicates that as a vessel is gradually stenosed the collateral circulation increases and supplies the compromised territory. In this particular case, the collateral circulation might have been provided by an increased blood flow via the circle of Willis, or more probably by an increased flow between the small arterial anastomoses of the cortex, since adjustments of flow via the circle of Willis are rapid, while adjustments mediated through cortical vessels may be delayed. The marked hyperemia of cortex occurring when the artery was released after 80 sec. of occlusion also suggests that local vasodilatation of cortical vessels had occurred.

In general, the studies in man reported here and elsewhere[11] reveal results comparable in most respects to those reported in experimental animals. Presumably because of the relatively greater size of the human brain, when supply of tissue oxygen fails to meet demand of tissue, the gradient of reduction of oxygen tension is more rapid and low levels may be reached in a matter of 8–12 sec. (Figs. 2 and 6). Conversely, when cerebral metabolism

is reduced by hypothermia in man (Fig. 7) there appears to be a relatively greater degree of protection against ischemia than is seen in the experimental animals.[11]

SUMMARY

Polarographic measurements of cerebral oxygen tension have been made in the neurosurgical operating room in cases of cerebrovascular disease and various forms of brain trauma. For purposes of comparison, measurements have been made in a 14-year-old girl without cerebrovascular disease. In 3 patients records were made while the internal or common carotid artery was occluded in the neck for the treatment of intracranial saccular aneurysm or vascular malformation. Polarographic measurements were correlated with the functional state of the cerebrum judged by clinical tests and the electroencephalogram. Repeated measurements of blood pressure were made in all cases; in some the cortical temperature, respirations and the electrocardiogram were also recorded concurrently.

The cortical oxygen tension appears to give a valid indication in man of local blood flow provided the blood pressure, oxygen content of the blood and cerebral metabolism remain constant. A severe reduction of blood pressure in the presence of thrombosis of the internal carotid artery results in a rapid reduction in availability of cortical oxygen except in areas where infarction and ischemic damage have reduced local metabolic consumption of oxygen.

Changes in oxygen content of the blood cause parallel changes in the oxygen tension of areas of cortex having an intact blood supply. Reduction of the local blood supply by vascular injury, thrombosis, hemorrhage or brain compression results in a corresponding delay in the changes in cortical oxygen tension induced by anoxic anoxia. In areas of blood clot and necrosis that are devoid of circulation, no change in the cortical oxygen tension occurs with anoxic anoxia.

The rate of reduction of cortical oxygen tension in the distribution of the middle cerebral artery, resulting from occlusion of the carotid artery in the neck, depends on the rate of local tissue metabolism and the degree of collateral circulation. If the collateral circulation is poor and the cerebral metabolism is active, the polarograph electrode will record a rapid reduction of oxygen tension to low levels within 8–12 sec., and the electroencephalogram will show slow activity, presumably because of local anoxia. Continued local anoxia results first in reversible functional impairment occurring within 30 to 60 sec.; if continued further infarction will occur.[3,9] If the collateral circulation is adequate, compensation usually begins within 45 sec. and the oxygen tension rises toward the steady state prior to release of the artery and functional impairment fails to result. If neuronal metabolism is lowered by hypothermia or by the damaging effects of ischemia or trauma, local hypoxia is less marked when the carotid artery is occluded. Release of the carotid artery after prolonged ischemia results in a localized super-

normal level of availability of oxygen which presumably is caused by local hyperemia.

The collateral cerebral blood flow may be increased by gradual occlusion of the internal carotid artery in small stages in those instances in which inadequacy is demonstrable by rapid closure of the vessel.

REFERENCES

1. Davies, P. W., and Brink, F., Jr. Direct measurement of brain oxygen concentrations with a platinum electrode. *Fed. Proc.*, 1942, *1:* 19.
2. Denny-Brown, D., Horenstein, S., and Fang, H. C. H. Cerebral infarction produced by venous distention. *J. Neuropath. exp. Neurol.*, 1956, *15:* 146–180.
3. Denny-Brown, D., and Meyer, J. S. The cerebral collateral circulation. II. The production of cerebral infarction by ischemic anoxia and the reversibility of early stages of the process. *Neurology.* (In press).
4. Kety, S. S., and Schmidt, C. F. The nitrous oxide method for the quantitative determination of cerebral blood flow in man: theory, procedure and normal values. *J. Clin. Invest.*, 1948, *27:* 476–483.
5. Meyer, J. S. Studies of cerebral circulation in brain injury. III. Cerebral contusion, laceration and brain stem injury. *Electroenceph. clin. Neurophysiol.*, 1956, *8:* 107–116.
6. Meyer, J. S. Studies of cerebral circulation in brain injury. IV. Ischemia and hypoxemia of the brain stem and respiratory center. *Electroenceph. clin. Neurophysiol.*, 1957, *9:* 83–100.
7. Meyer, J. S., and Denny-Brown, D. Studies of cerebral circulation in brain injury. I. Validity of combined local cerebral electropolarography, thermometry and steady potentials as an indicator of local circulatory and functional changes. *Electroenceph. clin. Neurophysiol.*, 1955, *7:* 511–528.
8. Meyer, J. S., and Denny-Brown, D. Studies of cerebral circulation in brain injury. II. Cerebral concussion. *Electroenceph. clin. Neurophysiol.*, 1955, *7:* 529–544.
9. Meyer, J. S., and Denny-Brown, D. The cerebral collateral circulation. I. Factors influencing collateral blood flow. *Neurology.* (In press)
10. Meyer, J. S., Fang, H. C., and Denny-Brown, D. Polarographic study of cerebral collateral circulation. *Arch. Neurol. Psychiat., Chicago*, 1954, *72:* 296–312.
11. Meyer, J. S., and Hunter, J. Effects of hypothermia on local blood flow and metabolism during cerebral ischemia and hypoxia. *J. Neurosurg.*, 1957, *14:* 210–227.
12. Meyer, J. S., Leiderman, H., and Denny-Brown, D. Electroencephalographic study of insufficiency of the basilar and carotid arteries in man. *Neurology*, 1956, *6:* 455–477.
13. Selverstone, B., and White, J. C. A new technique for gradual occlusion of the carotid artery. *Arch. Neurol. Psychiat., Chicago*, 1951, *66:* 246.

PHRENICOFACIAL NERVE ANASTOMOSIS FOR FACIAL PARALYSIS

ROBERT C. HARDY, M.D.,* GEORGE PERRET, M.D., AND RUSSELL MEYERS, M.D.

Division of Neurosurgery, State University of Iowa, Iowa City, Iowa

(Received for publication November 21, 1956)

ATTEMPTS to correct facial paralysis by anastomosing the distal portion of the facial nerve with a donor nerve began with the turn of the century. The first anastomosis of the spinal accessory to the facial nerve was performed by Faure[5] in 1898, and in 1903 Ballance and his associates[1] reported good results in similar anastomoses. Use of the hypoglossal nerve as the donor implant was reported almost simultaneously by Körte;[9] Kennedy[7] and Cushing[4] were among the first to report successful results of spinofacial anastomosis. The operative technic progressed from an end-to-side anastomosis to the correct end-to-end procedure. The glossopharyngeal nerve has also been used successfully by Watson-Williams.[15]

Recent reports of anastomosis of the facial nerve with the spinal accessory and hypoglossal nerves[3,10,12,14] have emphasized the inherent disadvantages of both procedures, including the undesirable mass movements, the failure of return of emotional and reflex responses, and the sometimes serious deficits secondary to surgical interruption of the donor nerve. Plastic procedures designed to restore facial symmetry have been helpful in cases in which repair of the nerve was not feasible.[2,13]

The facial nerve is so bound within the capsule of an acoustic neurinoma that removal of the intracranial portion of the nerve usually occurs with the total removal of the tumor. The use of either the spinal accessory or the hypoglossal nerve may not be warranted for anastomosis as a result of its involvement by the tumor or damage incidental to removal of the tumor. Our first case is that of a patient with facial paralysis following the total removal of an acoustic neurinoma treated by an anastomosis of the phrenic nerve with the facial nerve.

CASE REPORT

SUI #54–12720. The patient was a 44-year-old widow who complained of hearing roaring noises, deafness, diminished vision, difficulty in walking, nausea and vomiting. She had been aware of diminished hearing on the right side for many years, and she recalled that several years ago she had heard an almost continuous roaring noise for several months. For about 9 months she had noticed increasing difficulty in walking. For 3 months she had been bothered with episodes of nausea and vomiting of increasing frequency and severity. For 2 months she had had spells consisting

* Present address: 1233 Nix Professional Building, San Antonio, Texas.

of blindness and a strange sensation of numbness of the entire body lasting several minutes. For several weeks there had been increasing impairment of distant and near vision.

Examination. The patient walked with a wide-based unsteady gait. On Romberg testing she would fall to the right. There was drifting of the outstretched left upper extremity. The right eye could not be sustained in right lateral gaze. Papilledema was marked bilaterally. Moderate weakness of the right side of the face was evident. There was pronounced hypalgesia of the right side of the face, and scarcely any corneal sensation was present on this side. Bone conduction of the right ear was markedly diminished. The right heel-to-left knee performance was poorly executed.

Roentgenograms of the skull revealed pronounced enlargement of the right internal auditory meatus. Loss of function of both components of the right acoustic nerve was shown by audiometric and caloric tests.

Operation. On Oct. 29, 1954, a right suboccipital craniectomy was carried out and an encapsulated spherical tumor, approximately 3 cm. in diameter, was completely removed from the cerebellopontine angle. The inferolateral one-third of the cerebellar hemisphere was sacrificed to provide greater exposure. The clinical impression of acoustic neurinoma was confirmed microscopically.

Postoperative course was complicated by a *B. coli* meningitis, but after several days of intramuscular streptomycin and intravenous Terramycin therapy, the cerebrospinal fluid cultures were negative and the protein and sugar values approached normal limits. Gastric-tube feedings were necessary following operation, but after 4 weeks she was able to swallow adequately. The hoarse quality of speech also slowly improved. Her vision continued to deteriorate for 2 months, at the end of which time it was 2/60. There was no further loss of vision. She had a disfiguring facial palsy, and in addition there was wasting of the right side of the tongue, weakness in elevation of the right shoulder, and wasting of the corresponding trapezius and sternomastoid muscles.

A facial nerve anastomosis was desired, and in view of the significant damage to the spinal accessory and hypoglossal nerves, it was decided to use the phrenic nerve.

2nd Operation. A right phrenicofacial anastomosis was carried out on Dec. 30, 1954. The phrenic nerve was exposed through a transverse incision just above the clavicle. The nerve was followed as far inferiorly as possible before it was cut. It was transposed deep to the sternomastoid muscle to a wound near the angle of the jaw, where the trunk of the facial nerve was exposed. A small incision between the two wounds was made to facilitate the transposition. In order to gain necessary length, the contribution from the 5th cervical nerve was sacrificed. The phrenicofacial anastomosis was accomplished with epineural sutures of #00000 silk. Rotation of the head to the left of midposition was avoided by the use of a chin-shoulder adhesive strap for 4 weeks.

Course. On Mar. 13, 1955, 5 months after the craniectomy and 3 months after the phrenicofacial anastomosis, the patient's gait was improved, she swallowed more easily and her voice was normal. An iridocyclitis had developed, which responded favorably to treatment. There was marked flaccidity of the facial musculature of the right side.

Three months later, June 22, 1955, a return of muscle tone of the right side of the face was evident. In repose, a barely perceptible movement of the right side of the mouth was observed to coincide with each inspiration. With a deep inspiration the face was pulled to the right side and the right lid would close (Fig. 1B).

The patient and her family had been unaware of the significance of these changes. After only a few minutes' instruction, she was able to correlate the depth of inspiration with the extent of the smile to result in a symmetrical facies (Fig. 1C).

The patient was re-examined on Mar. 20, 1956 and again on Oct. 3, 1956, 15 and 23 months respectively following anastomosis. She had manifestly held the gains recorded previously. In repose her facies appeared nearly symmetrical and thus suggested progressive improvement in muscle tonus. However, she had not yet learned regularly to correlate the movements of self-directed breathing with the play of facial movements. On some occasions she succeeded and on others failed (forgot?) to activate the right side of the face with the left.

Whether this irregular performance was because of lack of sufficient

FIG. 1. Photographs taken June 22, 1955, 6 months after phrenicofacial anastomosis: (A) In repose. (B) With deep inspiration. (C) Simultaneous smile and inspiration.

motivation or more basic shortcomings of the patient's capacity to learn new patterns of behaviour can only be speculated upon.

During fluoroscopy and radiographic studies of the chest on Mar. 20, 1956 both leaves of the diaphragm moved synchronously through approximately the same ranges of excursion. This unanticipated finding has been noted in other connections. Thus, Klein[8] asserted that failure to obtain hemidiaphragmatic paralysis following phrenicotomy appeared in 20 to 30 per cent of his cases, a figure that agrees closely with Hegner's[6] earlier statement on the subject. The explanation proffered is that accessory contributions from sources in the thorax join the phrenic nerve below the cervicobrachial level.

Since our initial experience we have successfully accomplished phrenicofacial neurorrhaphy in 4 additional patients. As in the first case, 2 of these operations were performed after extirpations of acoustic neurinoma. In another patient, the operation followed removal of a meningioma from the

cerebellopontine angle and in a fourth, it was performed for hemifacial paralysis following a gunshot wound which had shattered the left facial nerve and styloid process.

A brief synopsis of the status of the 4 patients follows: In the case of meningioma of the cerebellopontine angle, left phrenicofacial neurorrhaphy was accomplished on Nov. 7, 1955, and 5 months later "voluntary" contractions were seen about the left mouth and cheek but not in the frontalis region. However, 7 months postoperatively, a return of movements in the left frontalis and musculature of the eyelid was noted. Fluoroscopic examination revealed the left diaphragm higher than its fellow, moving through a small arc of excursion and in unison with the leaf of the right side. Our second case of phrenicofacial neurorrhaphy following acoustic neurinoma was carried out on the right side on Dec. 6, 1955. At checkup examination 6 months later the patient already exhibited some "voluntary" motion around the right eye and corner of the mouth. One year later there was good, though incomplete, recovery of facial motion. At fluoroscopy this patient exhibited a paradoxical movement of the right leaf of the diaphragm. In our third case of acoustic neurinoma, phrenicofacial neurorrhaphy was performed on Nov. 27, 1956. As only $2\frac{1}{2}$ months have passed since this operation, we are not prepared to report meaningfully on the case. Finally, in the instance of the individual who suffered the gunshot wound, the first evidence of facial function was seen 6 months after the operation. He has continued to improve for several weeks thereafter.

We desire to report, in addition to the above cases, an unsuccessful attempt to accomplish phrenicofacial neurorrhaphy in 1 patient following removal of an acoustic neurinoma. In this instance it was not possible to accomplish suture of the nerves because we were unable to mobilize a sufficient portion of the phrenic nerve to permit approximation of its proximal end to the distal end of the facial nerve. The phrenic nerve in this case appeared upon anatomic dissection to arise completely from the 5th cervical nerve, in contrast to the usual circumstance in which it arises from the 3rd and/or 4th cervical nerves. It is likely that whenever this particular anatomic variation obtains it will compromise the surgeon's attempt to accomplish phrenicofacial anastomosis, unless he is prepared to pursue the trunk well into the mediastinum.

DISCUSSION

Although Cushing[4] suggested in 1903 that a motor branch of the cervical plexus might be used in facial nerve anastomosis and Lurje[11] mentioned the phrenic nerve as a potentially suitable implant into the peripheral ends of severed components of the brachial plexus, to our knowledge these are the first reported cases of phrenicofacial anastomosis.

The present data suggest that clinical evidence of regeneration of fibres within the facial nerve is as satisfactory following phrenicofacial as that following spinofacial and hypoglossofacial neurorrhaphy. If our impression in this regard should be supported as additional cases are accumulated in this

and other clinics, there would appear to be reasons for preferring the phrenicofacial to the more conventional procedures. In the instance of spinofacial neurorrhaphy, an asymmetry of the musculature of neck and shoulder must be accepted regularly and aching discomfort of the upper limb is a not infrequent consequence of the partial paralysis. In the instance of hypoglossofacial neurorrhaphy, a bothersome dysarthria and some disability in manipulating food within the mouth often attend hemiparalysis of the intrinsic muscles of the tongue. Phrenicofacial anastomosis avoids such consequences of visible atrophy, dysarthria and impairment of motor power. Even if it is likely, as apparently obtains in about 70 to 80 per cent of cases, that hemidiaphragmatic paralysis supervenes after such an operation, the patient's presenting appearance would in no way be compromised. Finally, the physiologic disability resulting from section of one phrenic nerve is virtually nil.

SUMMARY

A case is reported in which a phrenicofacial anastomosis was carried out for facial paralysis which followed the total removal of an acoustic neurinoma. The result 23 months later is considered fairly satisfactory. More recent experience with 5 additional cases is briefly presented.

REFERENCES

1. Ballance, C. A., Ballance, H. A., and Stewart, P. Remarks on the operative treatment of chronic facial palsy of peripheral origin. *Brit. med. J.*, 1903, *1:* 1009–1013.
2. Brown, J. B. The utilization of the temporal muscle and fascia in facial paralysis. *Trans. South. Surg. Ass.*, 1938, *51:* 352–359.
3. Coleman, C. C., and Walker, J. C. Technic of anastomosis of the branches of the facial nerve with the spinal accessory for facial paralysis. *Ann. Surg.*, 1950, *131:* 960–966.
4. Cushing, H. The surgical treatment of facial paralysis by nerve anastomosis. With the report of a successful case. *Ann. Surg.*, 1903, *37:* 641–659.
5. Faure, J.-L. Traitement chirurgical de la paralysie faciale par l'anastomose spino-faciale. *Rev. Chir., Paris*, 1898, *18:* 1098–1099.
6. Hegner, C. F. Surgery of pulmonary tuberculosis. In: Christopher, F. *A Textbook of Surgery.* Philadelphia & London: W. B. Saunders Co., 1950, 5th ed., 874–892 (see p. 882).
7. Kennedy, R. On the restoration of co-ordinated movements after nerve-crossing, with interchange of function of the cerebral cortical centres. *Philos. Trans.*, 1901, *194B:* 127–162 (see p. 152).
8. Klein, B. Phrenic interruption in the treatment of pulmonary tuberculosis. *Ill. med. J.*, 1938, *73:* 418–420.
9. Körte, W., and Bernhardt, M. Ein Fall von Nervenpfropfung: des Nervus facialis auf den Nervus hypoglossus. *Dtsch. med. Wschr.*, 1903, *29:* 293–295.
10. Love, J. G., and Cannon, B. W. Nerve anastomosis in the treatment of facial paralysis. Special consideration of the etiologic role of total removal of tumors of the acoustic nerve. *Arch. Surg., Chicago*, 1951, *62:* 379–390.
11. Lurje, A. Concerning surgical treatment of traumatic injury of the upper division of the brachial plexus (Erb's type). *Ann. Surg.*, 1948, *127:* 317–326.
12. McKenzie, K. G., and Alexander, E., Jr. Restoration of facial function by nerve anastomosis. *Ann. Surg.*, 1950, *132:* 411–415.
13. McLaughlin, C. R. Permanent facial paralysis. The rôle of surgical support. *Lancet*, 1952, *263:* 647–651.
14. Trumble, H. C. Nerve anastomosis in the treatment of facial paralysis. *Aust. N.Z. J. Surg.*, 1948, *17:* 297–299.
15. Watson-Williams, E. Glosso-pharyngeal facial nerve anastomosis. *Proc. roy. Soc. Med.*, 1927, *20:* 1439–1442.

A NEW TECHNIQUE FOR THE MICROSCOPIC EXAMINATION OF CEREBRAL VESSELS *IN VIVO**

SALAMON HAKIM, M.D., AND C. MILLER FISHER, M.D.

Neurology Service, Massachusetts General Hospital, and Department of Neurology-Neuropathology, Harvard Medical School, Boston, Massachusetts

(Received for publication December 13, 1956)

DURING the past century, microscopic studies of the cerebral vessels of living animals have been made by many investigators of whom Hill, Florey, Forbes, Wolff, Villaret and Cachera must be especially mentioned. Forbes'[1] introduction of the cranial-window technique made it possible to carry out observations under more physiological conditions, and the avoidance of secondary movements of the brain during respiration aided greatly both in the direct examination of the cerebral vessels and in the photographic recording of the vascular alterations. In general, the various authors reached approximately the same conclusions, namely, that the cerebral arteries are relatively inactive to neural and chemical stimuli in comparison with similar-sized vessels in other regions of the body.

In attempting to observe the state of the smaller vessels (15–50 μ) and measure their caliber accurately, we have utilized some of the recent improvements in microscopic and photographic equipment, and have developed a satisfactory technique in which the Leitz Ultrapak incident light illuminator is employed. In this apparatus (Figs. 1, 2, 3), illumination is introduced from the outside, transmitted through the objective and focused on the field under observation by means of a ring of condensers which surround the objective lens. The illumination is brilliant and color-photography is possible at magnifications of more than 240×. While the above procedure was being perfected, our principal goal in working in the field of experimental vascular physiology remained the development of a method whereby human vessels might be viewed microscopically in the operating room during craniotomy. This too has been achieved and it is now possible for the first time to take the Ultrapak apparatus to the operating theater and observe and photograph human cerebral vessels not only in their "normal" state but also under the influence of various stimuli. This technical advance is simply the result of using "Saran" paper—a plastic wrapping tissue—as a sterile drape over the exposed cortex. The Saran material, which is transparent, preserves the sterility of the operative field without interfering in any way with the microscopic examination. The present communication is a preliminary report of the method as it has been developed so far.

* This work was supported in part by Grant B-731(C) from the National Institutes of Health, United States Public Health Service.

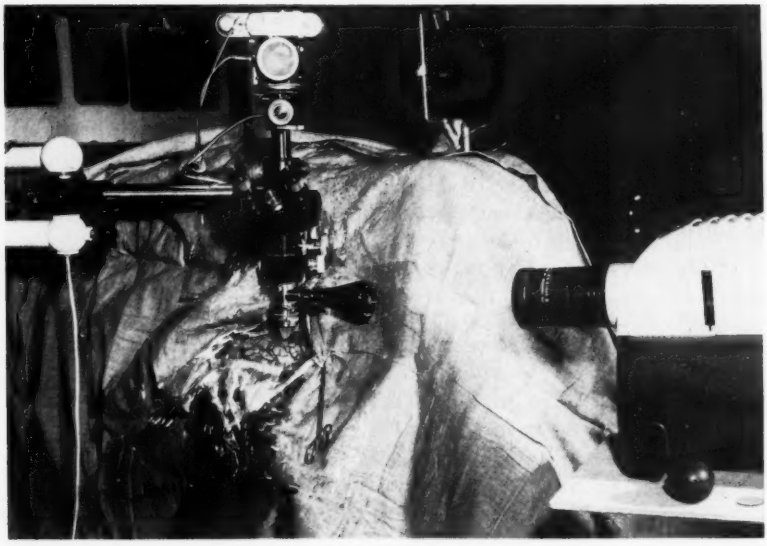


FIG. 1. Photograph of the assembled apparatus in the operating room.

The Use of the Ultrapak. Figs. 1 and 2 show the assembled apparatus in the operating room. The body of the Ortholux microscope is carried on the stand of a floor-model dissecting-microscope by means of a metal arm provided at

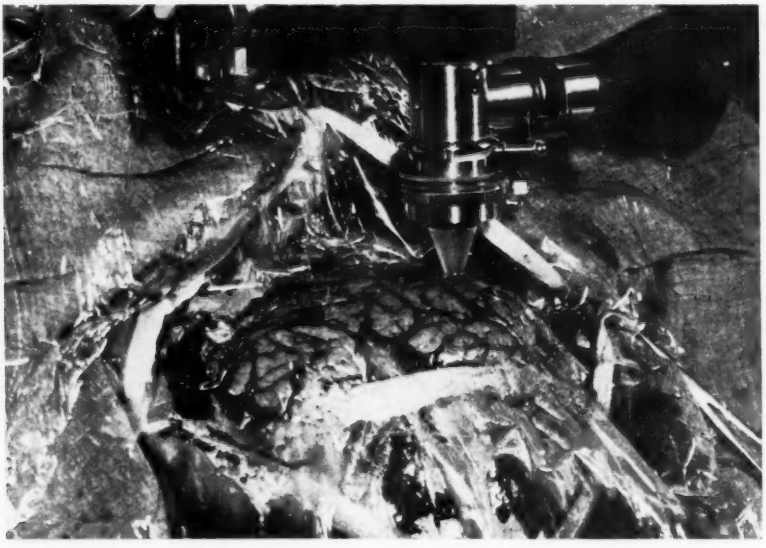


FIG. 2. Close-up photograph of surface of brain being examined.

its end with a special holder. In place of the revolving nosepiece of the microscope, a Leitz Ultrapak incident light illuminator is attached. Below this is inserted an Ultrapak objective which is available in several magnifications. In order to eliminate undesired surface reflections, "dipping cones" are used, a small amount of water or immersion oil filling the gap between the cone and the Saran over the surface of the brain. In our experiments, the cerebral cortex has been exposed by a routine craniotomy and the window technique has not been used. For animal work, when movement of the brain caused by respiration interferes with observations, the dipping cone may be slightly lowered so as to impinge lightly on the brain and hold the field steady. This can usually be done without compressing vessels. Immersion oil, because of its high viscosity, damps movements more effectively than saline. Satisfac-

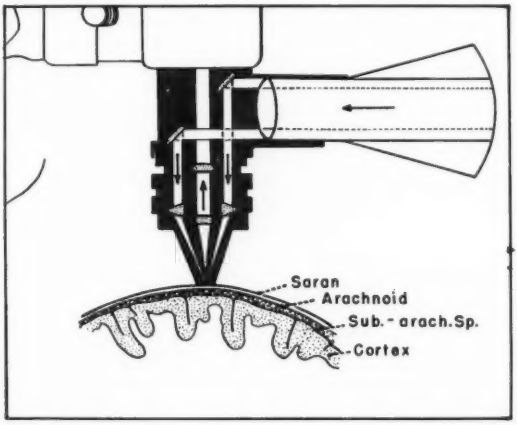


FIG. 3. Diagram of the optical system in the Leitz Ultrapak.

tory illumination has been achieved by using as the light source a 35 mm. projector carrying a 500 or 1,000 watt bulb. The position of the light is adjusted by trial and error until the illumination at the eyepiece of the microscope is maximum as determined by a photometer. This provides a bright field without warming the surface of the brain and thermocouple measurements have shown that the surface of the exposed brain remains slightly below body temperature. The Ultrapak apparatus is so constructed that color-filters can be introduced as desired. Provision is also made for the use of polarized light which abolishes glare and irregular reflection although this reduces the illumination considerably. For photographic purposes, a Leica camera has been used with microibso and reflex housing attachments. Satisfactory colored motion pictures have been obtained with a Bolex camera.

The Use of "Saran" Wrap. This material, a form of plastic, is thin, transparent and flexible. When applied to the brain, it tends to cling to the sur-

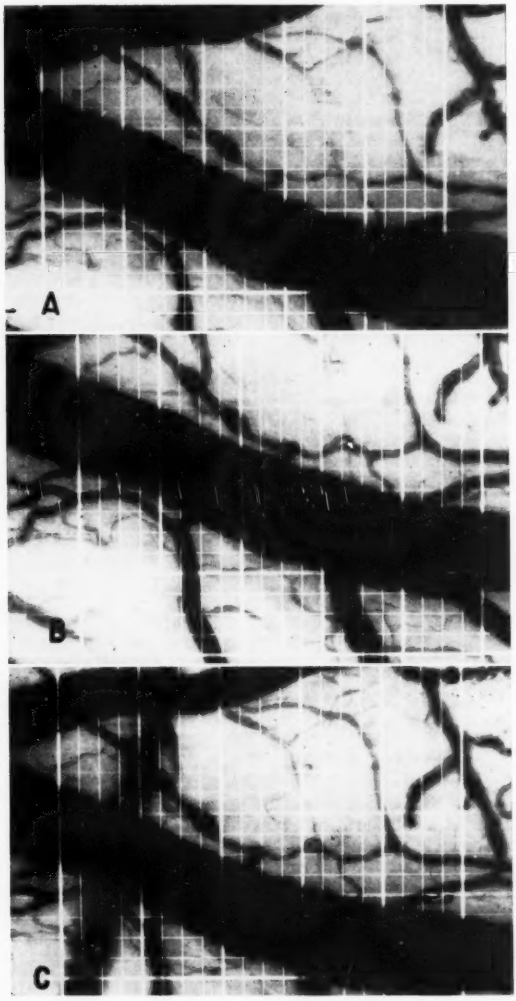


FIG. 4. Brain vessels of cat: (A) before topical Adrenalin 1:20,000; (B) 1 minute later; and (C) 2 minutes later. The arteries and veins are slightly wider in (B) and (C). This failure of vessels to constrict is in contrast with the reaction of arteries of the scalp, as seen in Fig. 5. The calibration grid is taken from a hemocytometer which has been photographed using the same optical system as used in the experiments. The negative is then saved and routinely superimposed (emulsion surface to emulsion) over the experimental negatives when the latter are being printed.

face and air-pockets usually do not form under it. According to the manufacturers,* it is highly impervious and we have found that liquid suspensions of bacteria when placed in contact with it do not permeate through in a

* Dow Chemical Corporation.

period of 48 hours. "Saran" does not not permit the passage of water, is strong, and withstands sterilization by autoclaving or immersion in 80 per cent alcohol. Autoclaving causes some shrinkage, and the latter method of sterilization is probably preferable. In the operating theater, the neurosurgeon, after reflecting the dura mater and completing the exposure of the pial surface, covers the entire sterile territory with an extra craniotomy drape, leaving exposed only the brain surface that is to be observed. He then covers this region with a large piece of sterile Saran paper and the field is ready for microscopic study. Chemical solutions can be applied to the surface under observation via a long, fine polyethylene tube, one end of which

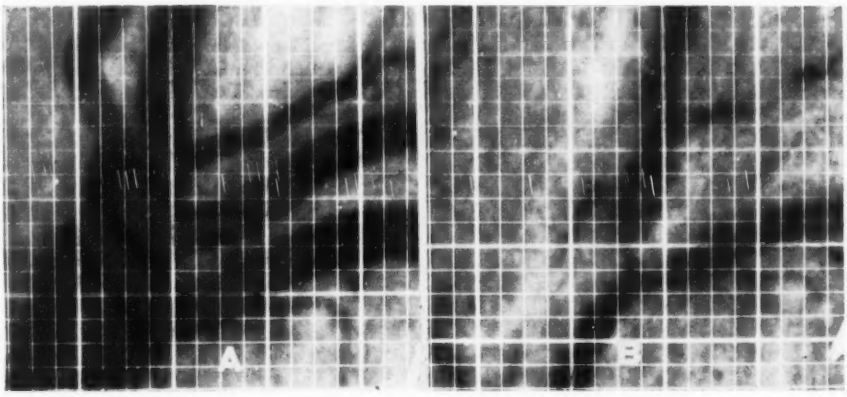


FIG. 5. Arteries of the scalp of a cat: (A) before Adrenalin 1:20,000; (B) 1 minute later.

has been arranged in position on the brain just before the Saran paper is put in place.

Preliminary Observations. By this method, arteries, veins and even capillaries on the surface of the brain can be viewed with unsurpassed clarity both in humans and animals. Moreover, accurate photography of vessels as small as 15 μ is not difficult. Only the subarachnoid and pial vessels can be seen, the dense opacity of the superficial layer of the cortex preventing observation of the circulation within the brain substance.

The flow of blood in arteries and veins presents a most striking spectacle. In animals, the arteries are brighter red in color than the veins and the two can be easily distinguished. Flow in veins is slower so that the mass of erythrocytes can be seen moving along, whereas in arteries the flow is usually so rapid that movement cannot be discerned. As though in compensation for this slower venous flow, the veins are larger and more numerous. The mass of blood in veins is usually arranged in several parallel streams. This streaming effect results from the fact that columns of blood entering the main vein from tributary vessels do not mix freely but tend to maintain their separate identity for some distance. The streams travel at different speeds

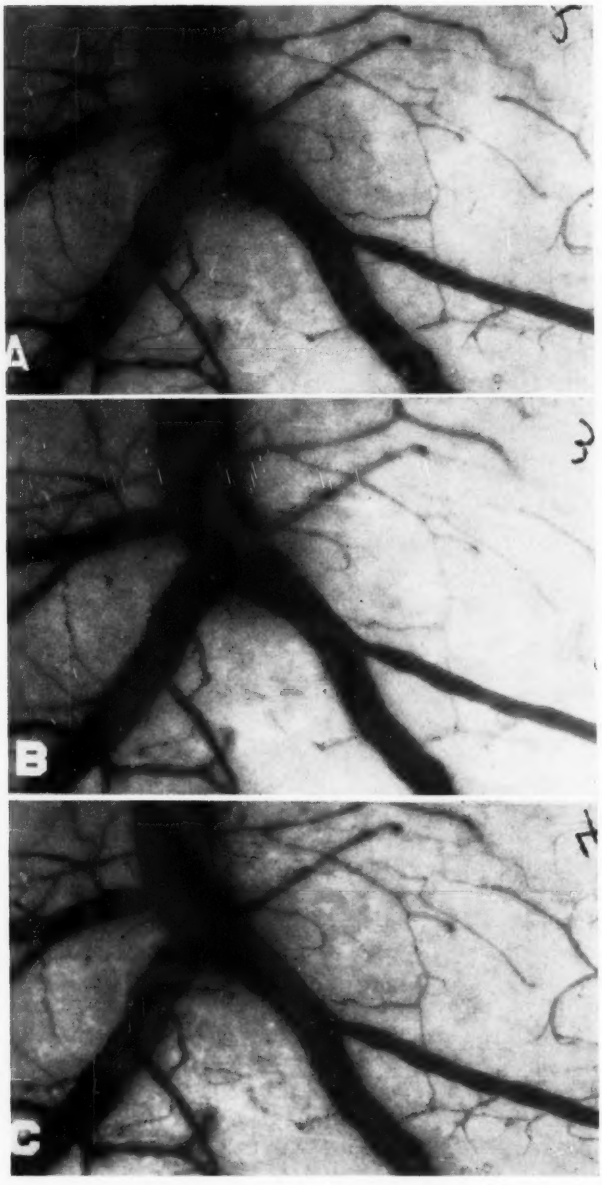


FIG. 6. Vessels of a human brain photographed at 50×: (A) before Adrenalin 1:20,000; (B) 1 minute later; (C) 2 minutes later.

and they also differ in color because of variations in the degree of reduction of their contained hemoglobin. Anastomotic channels connecting small pial arteries (20–50 μ) have been seen regularly. Whereas color photographs allow arteries and veins to be distinguished because of their difference in color, in black and white pictures this is not possible. In the latter, however, the veins can often be identified because of the presence of streaming.

Thus far, most of the physiological studies have been made in cats and rabbits, and the results are largely confirmatory of the conclusions of previous authors. It would appear that the small cerebral arteries of animals are

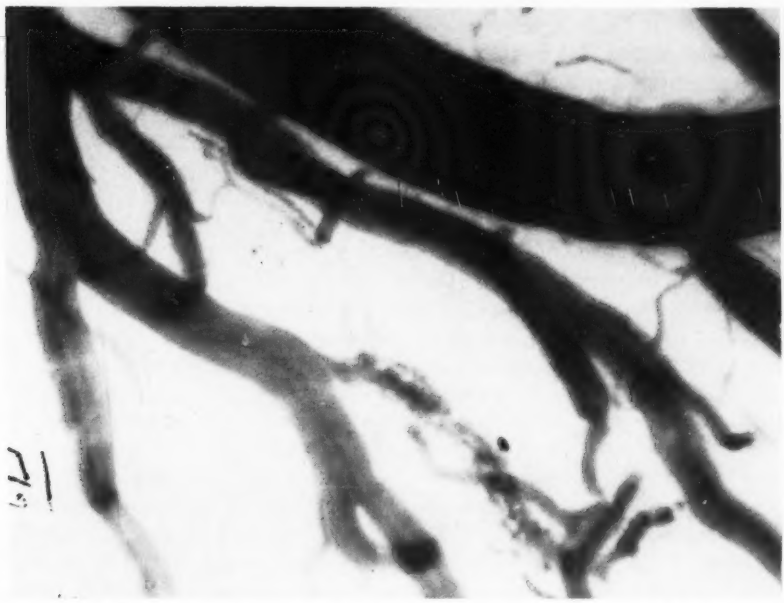


FIG. 7. Brain vessels of cat to illustrate venous "streaming" in vessels at left, below.

no more responsive than larger vessels as determined by investigators in the past, and indeed under Nembutal anesthesia we have not been able to record narrowing of arteries after the local application of Adrenalin 1:20,000. Neither has Noradrenalin had a measurable effect, and the same is true of stimulation of the ipsilateral cervical sympathetic chain.

In one series of experiments, we studied the effect on cerebral vessels of a mixture of alcohol and ethyl cellulose similar to that used for chemical pallidotomy. Within seconds of applying the material, widespread arrest of flow in arteries and veins occurred, illustrating the need to keep in mind the possibility that chemicals injected into restricted regions of the brain may produce unexpected widespread effects through occlusion of blood vessels that run through the injected zone to more distal parts.

In several experiments, it was found that by keeping the exposed cortex covered with Saran paper the brain remained perfectly healthy for as long as 12 to 14 hours. The vessels showed no pathological changes, their resting caliber did not alter and furthermore, brain swelling did not occur despite numerous chemical and physical manipulations. No doubt abnormal gaseous and aqueous changes in the superficial layers of the cortex were minimized.

Since it is our intention to limit this paper largely to a report of the methods used, a fuller account of our findings will not be presented at this time. Observations of the cerebral circulation and studies of the function of cerebral vessels in humans are being pursued currently.

It is possible that Saran paper will be useful in neurosurgery for covering the exposed cortex and for wrapping muscle tissue, and also that it will be helpful in carrying out microscopic observations of the circulation in other human organs and tissues.

REFERENCE

1. Forbes, H. S. The cerebral circulation. 1. Observation and measurement of pial vessels. *Arch. Neurol. Psychiat., Chicago*, 1928, *19:* 751–761.

THE USE OF RADIOACTIVE IODINATED SERUM ALBUMIN IN THE LOCALIZATION OF INTRACRANIAL LESIONS*

RICHARD B. RHODY, PH.D., AND GERALD R. NOWLIS, M.D.†
Radiation Research Laboratory, and Department of Surgery, State University of Iowa, Iowa City, Iowa

(Received for publication December 20, 1956)

IN RECENT years radioactive isotopes have been used increasingly in attempts to localize intracranial lesions.[2,4,7] These studies are performed during the preoperative work-up of a patient and afford ready correlation with other diagnostic procedures, such as angiography, electroencephalography, pneumoencephalography, and ventriculography, which are carried out during the same period.

It is thought that the radioactive materials reach a higher concentration at the site of pathology because of a "disruption" in the blood-brain barrier. The blood-brain barrier represents a selective permeability of blood vessels within the central nervous system.[1] This is probably resident either in the intima of the cerebral capillaries or in the sucker feet of astrocytes and excludes selectively many molecules and electrolytes from the nervous system while allowing the usual transcapillary exchange of metabolites to occur.

In efforts made to localize intracranial pathology with radioactive materials, two more or less independent lines of endeavor have evolved. The first of these, and the one with which we are concerned here, utilizes the differential "pooling" of such radioactive substances as diiodofluorescein (DIF) and iodinated human serum albumin (RISA) by tissues within the cranial cavity that possess a capillary intima with different properties of permeability than the surrounding "normal" capillaries which maintain the blood-brain barrier. External radiation detectors are used to localize these tissues. RISA was used exclusively in this series.

The other method of radioactive survey is one with which we have had no experience. It involves the injection of such substances as P-32 preoperatively, and the use of a Geiger-Müller counter at the operating table after a bone flap has been turned. This presupposes that the general area of the brain in which the tumor resides is known. Thus, if one cannot identify neoplastic tissue grossly, the probe-counter is inserted into the cerebral substance and mapping is performed. An increased uptake in an area suggests the presence of neoplasia. In attempting total extirpation it enhances the possibility of identifying residual nests of tumor which might otherwise be missed.[5,6]

* Supported in part by the American Cancer Society, Institutional Grant, 1955–1956.
† Present address: The Mason Clinic, Seattle, Washington.

METHOD

Our procedure entailed the intravenous administration of 500 microcuries of RISA in adults and proportionately smaller amounts in children. This amount of radioactivity gave relatively high counting rates with scintillation counters. No deleterious effects were observed in any of our patients. All patients were premedicated with Lugol's solution to protect the thyroid from the radioactive iodine liberated in the metabolism of RISA. It was found that a period of between 20 and 30 hours after injection represented the optimal time for localization studies.

Scintillation counters consisting of NaI (Tl) crystals attached to the requisite photomultiplier tubes, voltage supplies and recorders have been used as detectors throughout these studies. Scalars were used to record the total number of counts in a unit of time. Several methods of scanning with these detectors have been tried. The preliminary work used a skull filled with rice and sawdust to simulate a brain. This phantom brain was made uniformly radioactive by soaking in an I-131 solution. "Tumors" of various sizes and specific activities were placed in different positions within the cranium and localization was attempted. A few of the methods of scanning that have been used will be discussed briefly, with an evaluation of their worth.

One of the first attempts at localization in this laboratory used a single counter which moved about the head in a circular manner. The scans were made in both spherical and cylindrical coordinates with the detector kept at a fixed distance from the center of the head or an axis passing through this center. This method relies on a comparison of the observed isocount contours with the contours obtained from a "normal" scan. This procedure was discarded because of the time consumed in a scan and because it was necessary for the patient's head to be held erect, a requirement that often cannot be met.

The next course of study utilized a pair of detectors which were fixed so that the crystals faced each other. With the median plane of the head placed symmetrically between the counters, the scan was made in a rectangular grid fashion. The tumor depth was determined from the difference in reading of the two counters. This method gave good results with a "brain to tumor" activity ratio as low as 5:1, unless it were in the center of the cavity. Although this procedure showed considerable promise it was discarded as many patients could not remain in a fixed position for the length of time that was necessary to complete a survey.

Because of the varied conditions under which patients have been surveyed, it has been found that the most flexible arrangement is the most satisfactory. In all of the cases reported here the counter was mounted on a flexible arm so that it could be held perpendicular to the head. In a few instances the detector was kept at a fixed distance, but usually it was in contact with the head.

The patient was made to don a tight-fitting cap upon which numbers were

placed symmetrically. This is shown in Fig. 1. Usually 50 to 60 positions were counted: i.e., 25 to 30 on each side of the head. The comparison of paired positions coupled with a "normal" pattern from a control made localization of radioactive concentrations possible. A $1\frac{1}{2}\times1$ inch NaI (Tl) crystal was shielded from the side by $\frac{5}{8}$ inch of lead, and from the front by $\frac{3}{4}$ inch of lead. The front shield contained a $\frac{3}{4}$ inch aperture, which allowed the crystal to "see" a half-angle of 26 degrees. The net counting rates were usually much greater than 3000 counts per minute. Because of these high counting rates a counting time as short as 20 seconds was sometimes used with a probable counting error of less than 3 per cent. To test the reliability of

FIG. 1. Example of the scanning technique used. The tight-fitting hat worn by the patient has numbers on it which define specific areas of the head. The counter and patient's head are usually supported by hand to prevent movement during a counting period.

our procedure, at least two surveys were always made; any reading that did not reproduce to within 10 per cent was discounted. No significance was attached to readings that did not show a lateral differentiation of 10 per cent unless the over-all pattern deviated markedly from a "normal" pattern. If the differentiation was less than 10 per cent, it was almost invariably much lower, that is, in the neighborhood of 3 to 6 per cent. This is true of not only the negative cases (those in which space-taking lesions were not found), but also in the instances in which a neoplasm was missed by RISA studies.

MATERIAL AND RESULTS

Approximately 70 patients were studied in the course of a 1-year period, using the procedure as outlined above. Only 50 of these have been included in the following statistical analysis, the reason for this being that insufficient

follow-up data are available on all of the patients to allow for meaningful interpretation. No attempt has been made at preferential selection, our only criteria for inclusion in this analysis being histological verification of tumor type either at operation or postmortem examination, or, in the case of other disease entities, indisputable evidence of their presence by adequate follow-up studies. The exceptional circumstances to this are two: first, a deep-seated hemispheral angioma which was demonstrated by angiography but not operated upon because of the patient's intact neurological status; and second, a tumor in the posterior portion of the 3rd ventricle having a clinical course consistent with pinealoma as well as the usual salutary response to roentgen-ray therapy.

Table 1 is a breakdown of the cases studied. The criterion as to whether the scintillation counter studies were "right" or "wrong" was based upon their ability to predict the presence or absence of a presumed space-taking intracranial lesion. From the pathophysiologic standpoint, this is possibly an erroneous presumption; in fact, one may even say that it probably is. To divide arbitrarily all intracranial lesions into those that displace cerebral tissue and those that do not is certainly not reasonable if one is to think dynamically in terms of the blood-brain barrier. For instance, where would one "pigeon-hole" the edema that is at times consequent upon cerebral thrombosis? We do not ordinarily think of an infarcted area as being a "space-consuming" lesion, and yet it is sometimes precisely that, even producing a massive shift of the ventricular system from one side to the other.

Our only justification for this artificial separation is that with the crude techniques available for differentiation of intracranial lesions, it is useful in that it helps pragmatically to decide whether a case is "surgical" or "non-surgical." Even this is fraught with pitfalls, however, in that aneurysms, which usually are not large enough to fall into the category of "mass lesion," are being more and more recognized as surgical lesions. The fact remains, however, that the great bulk of a neurologist's and neurosurgeon's referrals confront him with the questions: (1) Is this a mass lesion? (2) If so, where is it? Our results in using RISA are analysed in terms of pathological diagnoses in Table 1. The following are comments which would appear to be appropriate at this point.

Among the supratentorial gliomas, the 2 that were missed were both deep within the hemisphere and close to the mid line. Both were classed as grade IV astrocytomas. This might indicate that the cellular type among the gliomas is not especially related to their pooling of radioactive materials; or again, it might mean simply that the neoplasm was too far removed from the scalp to be detected.

The pontine gliomas, likewise, gave no hint of their presence. In dealing with the cystic cerebellar astrocytomas it was interesting to see that in both cases the uptake was somewhat greater (5 per cent) in the uninvolved cerebellar hemisphere as compared with the hemisphere that was the site of the tumor. After the diagnosis had been established in these cases, our impression

was that the cyst in a cerebellar astrocytoma may diminish the uptake of RISA on that side.

The same may be said of 2 supratentorial cysts which ultimately came to operation. One of these was a leptomeningeal cyst in the Sylvian fissure, the other a degenerated caseous lesion also in the posterior portion of the Sylvian fissure, the etiology of which was never determined, in spite of numerous cultures, immunologic studies, and varied stains obtained on the

TABLE 1

Breakdown of cases studied and results of localization experiments

Diagnosis	Number of Cases	Lesion Localized by RISA Study	Lesion not Localized by RISA Study
1. Glioma (Supratentorial)	11	9	2
a. Astrocytoma	9	7	2
b. Oligodendroglioma	1	1	
c. Ependymoblastoma	1	1	
2. Glioma (brain stem)	3		3
3. Glioma (cystic cerebellar)	2		2
4. Meningioma (supratentorial)	1	1	
5. Meningioma (infratentorial)	1	1	
6. Acoustic neurinoma	2	2	
7. Arteriovenous malformation	2	1	1
8. Metastatic neoplasm (supratentorial)	3	3	
9. Metastatic neoplasm (cerebellar)	2		2
10. Pinealoma	1		1
11. Subdural hematoma (bilateral)	1		1
12. Cysts	2		2
13. Diffuse cerebrovascular disease	6	6	
14. Cerebral thrombosis	3	3	
15. Congential aneurysm with intracerebral clot	1	1	
16. Cortical thrombophlebitis	2	2	
17. Basal arachnoiditis	1	1	
18. Arnold-Chiari malformation	1	1	
19. Controls	5	5	
Total	50	36	14

tissue removed at surgery. Both of these were avascular and had displaced cerebral tissue mesially.

There are 2 meningiomas in our series. One of these was a pterional lesion which had grown extracranially and presented as a tumor mass just above the zygoma. The most striking uptake was obtained in this case, there being a 30 per cent difference between readings taken over the area where only skin and subcutaneous tissue separated the tumor from the counter, and those taken over the same area on the opposite side of the head. The other meningioma was located in the left cerebellopontine angle. This was not a highly vascularized lesion, yet the RISA studies pointed unequivocally to this site preoperatively.

Two acoustic neurinomas were surveyed and accurately localized preoperatively. Both were large vascular neoplasms.

The metastatic tumors presented a confused picture. The 3 supratentorial lesions (1 epidermoid carcinoma of the cervix, 2 adenocarcinomas of the breast) were located precisely at the area where the scintillation counter showed the highest uptake. However, neither of the cerebellar metastatic deposits (1 epidermoid carcinoma of the lung, and 1 adenocarcinoma of the breast) were detected by this means.

One patient in the series had bilateral subdural hematomas. Our studies gave no hint of their presence. They were of approximately equal size, each one containing about 100 cc. of fluid. This case pointed up the need for a scale of reference so that one could, if necessary, compare the relative strength of readings over the various parts of the cranium with one or more points over the extremities, or another part of the body. This might then allow one to appreciate that the counts taken over the convexities were greater or lesser than anticipated, and thus to suspect a bilateral lesion. An attempt was made to compare the uptake of the frontal, temporal and parietal areas with the occipital and suboccipital regions in this case, utilizing the percentage difference arrived at with that found in controls. Here again, nothing of significance was uncovered.

No differential uptake was noted in patients with diffuse cerebral vascular disease nor in those who had cerebral thromboses with definite localizing neurological signs. Likewise, increased uptake was not found in cortical thrombophlebitis. One case of Arnold-Chiari malformation with internal hydrocephalus in a 5-year-old girl was studied, but again a differential uptake was not found. Five controls (normals) failed to show a difference in readings taken over the two sides of the head.

In the entire series only 1 "false-positive" was encountered. This was in a 55-year-old woman who had had a radical mastectomy some years previously for adenocarcinoma of the breast. She had subsequently become psychotic. When first seen there was unmistakable evidence of a lesion in the left cerebellar hemisphere. A RISA scan pointed to pathology in the right anterior frontal region, but ventriculography confirmed the presence of a space-taking lesion in the posterior fossa. At operation a metastatic nodule was enucleated from the left cerebellar hemisphere. The patient's papilledema subsided, her gait and co-ordination improved, but the psychosis persisted. She died some months later in another hospital, and unfortunately postmortem examination was not permitted. The possibility exists that this patient did indeed have another metastatic deposit in the frontal region which was not large enough to be demonstrated by ventriculography. Hence, this single "false-positive" may have been spurious.

In each of the cases in which accurate localization was accomplished by means of RISA, the highest uptake was invariably over the epicenter of the neoplasm. One of the authors (G.R.N.) was the surgeon in most of the cases reported herein, and entered into the experimental situation with some prejudice, having seen and examined the patient previously. The other

author (R.B.R.) was at all times the member of the team who positioned the scintillation counter and who made the extrapolations as to where the lesion would be on the basis of the radioactive uptake; in all instances the latter was kept ignorant of clinical findings that might localize the site of pathology. We were impressed particularly with the unequivocal nature of the results in those cases in which the lesions were correctly localized. This serves to fortify the opinion expressed by Farris *et al.*[3] that they were willing to attribute considerable significance to a "positive" (localizing) scan, but that in no way did a "negative" (nonlocalizing) scan exclude a space-taking lesion.

Because of the relatively small number of cases studied, it is impossible to compare the efficacy of the RISA technique with other procedures used in localizing intracranial pathology. Statistics based on 50 cases might well be misleading rather than enlightening. After some experience with the method, however, it is difficult to refrain from adopting certain attitudes, tentative though they may be.

We have felt that the temporal factor is a distinct disadvantage of this method. The institution in which this study was done serves a large referring area, and many of the patients who are sent for definitive diagnostic and therapeutic measures represent emergency situations which must be dealt with immediately. This applies not only to trauma but to intracranial neoplasms that have decompensated and require definitive therapeutic measures within a few hours following admission. It is certainly not justifiable to subject an already comatose patient or one whose level of responsiveness is deteriorating to a delay of 24 hours for the purpose of completing radioactive iodine studies, when angiography or ventriculography may make the diagnosis in one hour.

Perhaps the greatest advantage of the RISA scan is that it is entirely atraumatic to the patient. One need not insert cannulae through cerebral tissue nor upset the equilibrium of the cerebrospinal fluid with its frequently elevated pressure and precarious balance. Nor does one run the risk of disturbing the tone of the intracranial vascular tree as may happen with angiography. These facts, coupled with avoidance of an additional anesthetic induction, are cogent considerations to be taken into account in an evaluation of the method. In general, it has been our feeling that radioactive scans with RISA, taken by themselves, are less helpful than cerebral angiography, and/or gas contrast studies, but that they are distinctly more accurate in localizing a mass than is the electroencephalogram.

In dealing with the material at hand, one cannot help but idealize the qualities desirable in any technique that attempts to locate space-taking intracranial pathology. Among others, these merit particular notice: (1) rapidity of concentration in the pathological area; (2) minimum toxicity or other hazard to the patient; (3) lack of expensive equipment; and (4) simplicity of the test itself.

In searching for such a method it would seem that an agent permeable to the blood-brain barrier in normal cerebral substance, but not to the area

in which pathology exists, would be one useful approach. It is perhaps expecting too much of a method that it should concentrate enough of the telltale substance—radioactive or otherwise—in such varying morbid lesions as leptomeningeal cyst, astrocytoma, and intracerebral hematoma. To search for such a material is to disregard other basic concepts in anatomy, physiology and pathology. Indeed, because of the diverse nature of material with which a neurosurgeon must work, it may be that in the future we shall learn the proper combination of diagnostic tests to perform in any given case. This would constitute an acknowledgment of the fact that no one test gives us dependable answers in all cases.

We are unable to enter into any discussion of the reasons for the apparent preferential take-up of RISA in certain of these cases because our studies have not delved into the basic physicochemical mechanisms presumed to be at work. Relative vascularity is in some ways an attractive possibility, but clearly cannot be the whole answer. For instance, the 3 supratentorial metastatic tumors enucleated at operation were all poorly vascularized, yet were accurately localized preoperatively. This subject must be left for elucidation in the future.

SUMMARY

Fifty patients have been scanned with the scintillation counter following the intravenous injection of radioactive iodinated human serum albumin. Forty-five of these were subsequently proved to have intracranial pathology. Three volunteers without symptoms or signs of central nervous system disease were included, as were 2 patients with signs and symptoms that superficially suggested neurological disease, but who were later diagnosed as suffering from neuroses. These latter have been termed "normals" or "controls." The criterion of successful localization depended entirely upon the ability to predict the presence of a space-taking intracranial lesion. Statistics and histological diagnoses have been tabulated. This method appears worthy of further investigation. In the authors' hands, at least, it is not yet reliable enough to use to the exclusion of other standard diagnostic tests. The limits of its clinical applicability must be more clearly defined.

REFERENCES

1. Bakay, L. The blood-brain barrier with special regard to the use of radioactive isotopes. *Springfield, Ill.: Charles C Thomas*, 1956, xii, 154 pp.
2. Dunbar, H. S., and Ray, B. S. Localization of brain tumors and other intracranial lesions with radioactive iodinated human serum albumin. *Surg. Gynec. Obstet.*, 1954, *98:* 433–436.
3. Farris, R. G., Pantek, H. R., and Kahn, E. A. The clinical use of radioactive isotopes in the diagnosis and localization of brain tumors. *J. Mich. St. med. Soc.*, 1955, *54:* 825–827.
4. Moore, G. E. Diagnosis and localization of brain tumors. A clinical and experimental study employing fluorescent and radioactive tracer methods. *Springfield, Ill.: Charles C Thomas*, 1953, xxiii, 221 pp.
5. Schneider, R. C., Pantek, H., Freeman, D. G., and Farris, R. G. Radioactive phosphorus in the localization of the brain tumors. *J. Mich. St. med. Soc.*, 1955, *54:* 434–444.
6. Selverstone, B., Solomon, A. K., and Sweet, W. H. Location of brain tumors by means of radioactive phosphorus. *J. Amer. med. Ass.*, 1949, *140:* 277–278.
7. Sweet, W. H., and Brownell, G. L. Localization of intracranial lesions by scanning with positron-emitting arsenic. *J. Amer. med. Ass.*, 1955, *157:* 1183–1188.

GRAPHIC INTERPRETATION OF ISOTOPE LOCALIZATION OF INTRACRANIAL LESIONS*

SHELLEY N. CHOU, M.D.,† AND LYLE A. FRENCH, M.D.

Division of Neurosurgery, Department of Surgery, University of Minnesota Medical School, Minneapolis, Minnesota

(Received for publication December 21, 1956)

THIS report presents a new method of interpretation of data obtained by isotope encephalometry. The method, graphic analysis, makes possible more accuracy in localization of mid-line and deeply seated lesions and also helps to prevent misinterpretation of the data used in localizing cortical and subcortical lesions. This is accomplished by comparing counts of radioactivity obtained over specific areas of the two sides of the head with an established normal range for those sites.

In a normal individual there is a fairly even distribution of radioactivity in the brain after injection of a radioactive tracer substance. In the presence of an intracranial lesion in which the uptake of the tracer is either greater or less than the normal, an asymmetry in the counts recorded from comparable areas of the two sides of the head may be present. In the usual method of isotope interpretation reliance is placed upon an asymmetry of approximately 10 per cent. Obviously the most ideal situation for localization by asymmetry is one in which the lesion is in the cortex or immediately below the cortex. Such a lesion would be extremely close to the detector on one side and, therefore, yield an unequivocal focus. For lesions in the mid line the prospect is entirely different because any increased radioactivity in the lesion would be distributed equally to both sides of the cranium with no apparent asymmetry of activity. Conversely, benign cystic lesions with low uptake of tracer, especially if located peripherally in the brain, might afford an incorrect localization as a focus of increased activity on the opposite side. For these reasons the method of interpretation by graphic analysis was devised. The technique is based upon a graphic presentation of the relationship of specific activity encountered between each position in a given case as compared with that in a series of normal cases. Thus, disturbance of a normal relationship in the form of either a bilateral increase or a unilateral decrease of activity can be detected.

ESTABLISHMENT OF A "NORMAL GRAPH"

An analysis was made of 27 isotope encephalometric records that were considered normal. All 27 patients had been given a dose of radioactive

* This work was supported in part by research grants from the U. S. Public Health Service, B-665(C), and U. S. Atomic Energy Commission, AT(11-1)-285.

† Present address: 60 South Fourth Street, East, Salt Lake City, Utah.

iodinated serum albumin (RISA) in the amount of 5 μc./kg. Measurements of the routine 24 cranial positions were made 24 hours later. An arithmetical mean of the total counts as well as that for each position was obtained. A graph was made by plotting the specific activity for each position against cranial positions arbitrarily arranged numerically in series (Fig. 1). This graph presents the following features:

A) There are four high peaks of activity corresponding to positions 2, 5, 8, and 11. They are basally situated positions where thick layers of muscles and soft tissue are present.

B) Lowest activity is over positions 1, 6, and 9. These are areas of the cranium where very little soft-tissue covering is present.

C) Positions 4, 7, and 10 over the vertex are isometric. They are, as a

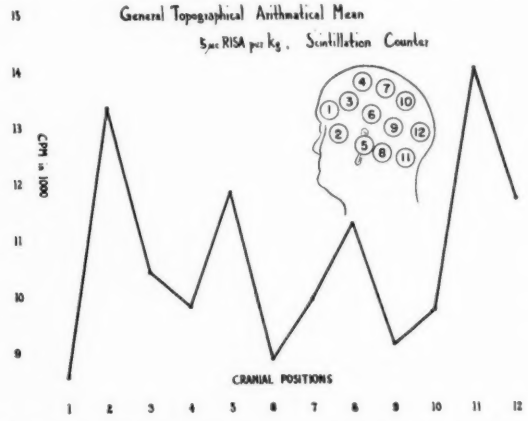


FIG. 1. This graph shows specific radioactivity for each of the designated cranial positions. The patient is given a uniform dosage of RISA.

group, higher than positions 1, 6, and 9 because the background is increased when the detector is positioned vertically against the head.

D) As these positions were calculated out individually in the normal series, they represent the arithmetical mean of each position. The symmetry of activity as measured over the corresponding positions of both sides is apparent. This is true also with respect to the variation from the mean of any case in the series of 27 "normals."

E) If the graph is separated into three zones, much like those in the colloidal gold curve for examination of cerebrospinal fluid, and a normal variation of each zone is studied, it is seen that the last zone corresponding to the occipital areas has the widest range of variation. The first and middle zones have equally little variation from the normal. This is again because of the variation in the amount of soft tissue over different positions in general and of that over the occipital region in particular. Therefore in any

given case, a slight deviation from the normal of the last zone does not carry as much significance as that of either the first or middle zone.

In order to magnify any abnormality present, this general topographic graph can be separated into several graphs describing planes, either coronal or sagittal, through the head (Fig. 2). In this manner, a total of six additional graphs can be obtained, one representing each plane (Figs. 3 and 4). Fig. 3 is a graph representing the activity pattern running from the right frontal position, along the vertex backwards to the right occiput and then forward through the left occiput to the left frontal position. This would include positions R1, R4, R7, R10, R12, R11, L11, L12, L10, L7, L4, L1 in sequence. This is called the vertex frontal-occipital-frontal graph. The next graph in Fig. 3 represents activity at the next plane, covering on both sides the intermediate between the vertex and the base of the skull (intermediate frontal-occipital-frontal graph). The third, representing the basal

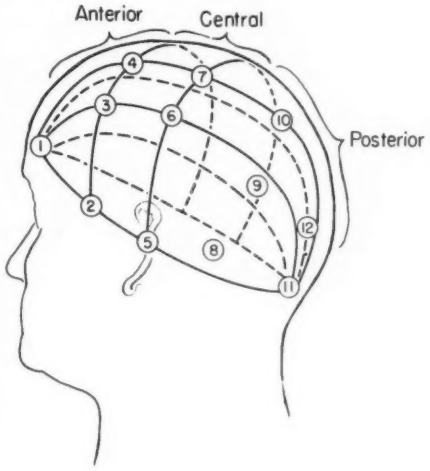


FIG. 2. Drawing of the skull to show the various planes used. There are three coronal (anterior, central, posterior) and three sagittal (vertex, intermediate, and basal) planes. (See text.)

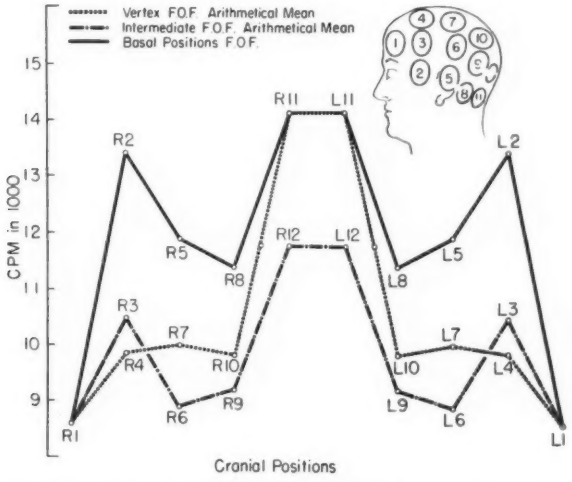


FIG. 3. Composite of graphs showing activity pattern in sagittal planes (vertex, intermediate, and basal). F.O.F. = frontal-occipital-frontal.

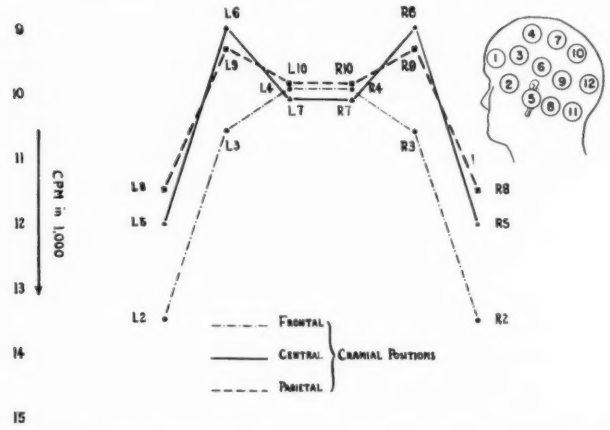


FIG. 4. Composite of graphs showing activity pattern in coronal planes (frontal, central, parietal).

positions, is called the basal frontal-occipital-frontal graph. The coronal planes, as represented in Fig. 4, are labelled the frontal, central, and parietal views and are constructed differently in that the lowest point in the curve indicates the highest activity. This is done to bring the curves more anatomically oriented so that when studied one can readily transform the planes into the corresponding anatomical locations.

There is a variation in absolute counting level for any given position of

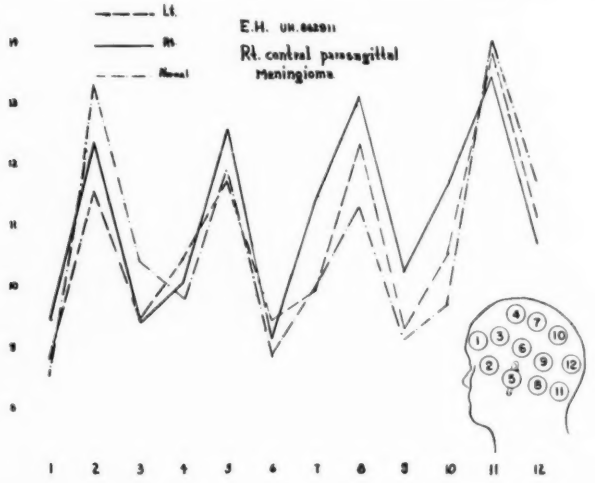


FIG. 5. *Case 1.* Graphic analysis (right central parasagittal meningioma). There is a slight elevation (increased counts per minute) on the right in the middle zone. There is also some elevation compared to the normal on the left, indicating a rather central (parasagittal or falx) location of the lesion.

as much as 40 per cent in different individuals having received the same dose of tracer substance on a μc./kg. basis. It is necessary, therefore, to convert the counting level of cases under study to that of the normal so that better comparison of the graphs can be obtained. This is done by converting the counts per unit time of each position under study with a factor which is the ratio of the total normal counts and the total counts of a case under study. For instance, if the total counts of a case under study is 130,000 then the conversion factor is 2 (260,000)/(130,000), 260,000 being the arithmetical mean of the series of 27 normals analyzed.

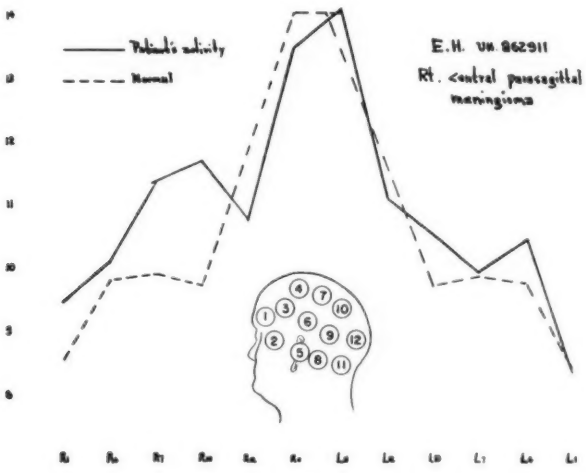


FIG. 6. *Case 1.* Sectional analysis graph (vertex frontal-occipital-frontal plane) demonstrating a definitely increased isotope count in the right parasagittal area.

ILLUSTRATIVE CASES

It seemed advisable to present first a case in which the lesion was clearly located by both the routine differential analysis technique and the graphic analysis technique.

Case 1. E.H., UH 862911. Meningioma, right parasagittal. Fig. 5 presents the graphs of this case. There is an apparent elevation of the middle zone, representing increased activity in the right central area. On further study, one sees that positions 7 and 10 on the right are markedly elevated as compared with those of the normal and that of position 4 of the unknown. Normally, positions 4, 7 and 10 are isometric, which is a very consistent finding. The elevation of activities found in these positions is definitely abnormal. Now if a sectional analysis is made, this abnormality becomes much more evident (Fig. 6).

Case 2. E.G., UH 862346. Brain abscess, left frontal. The general graph representing activity on the left in Fig. 7 shows a marked shift of activity upward in the

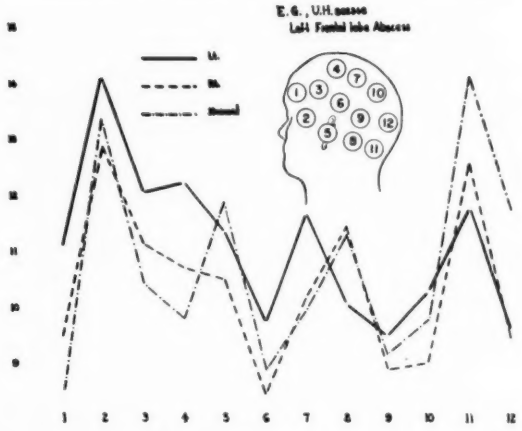


FIG. 7. *Case 2.* Graphic analysis (general graph). There is an upward shift of activity in the left frontal area suggestive of the lesion.

1st and 2nd zones. The elevation of activity in the left frontal area is unequivocal Here again position 4 is way out of its usual level compared with positions 7 and 10 A sectional analysis shown in Fig. 8 magnifies the abnormalities present.

Case 3. H.J., UH 829441. Glioblastoma multiforme, right frontal, extending across the mid line to the left. The original site of the tumor in the right frontal region had become partially necrotic with cystic loculations. On routine study with the isotope technique, a false focus was obtained on the left side.* By graphic analysis, however, it is clearly shown that there was abnormally increased activity in

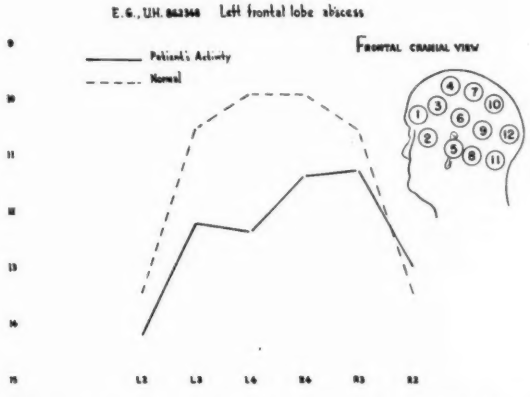


FIG. 8. *Case 2.* Sectional (coronal) analysis graph magnifies the alteration in counts from the normal curve.

* This case was reported by Peyton *et al.* in *J. Neurosurg.*, 1952, *9:* 432–442 (see Case 5).

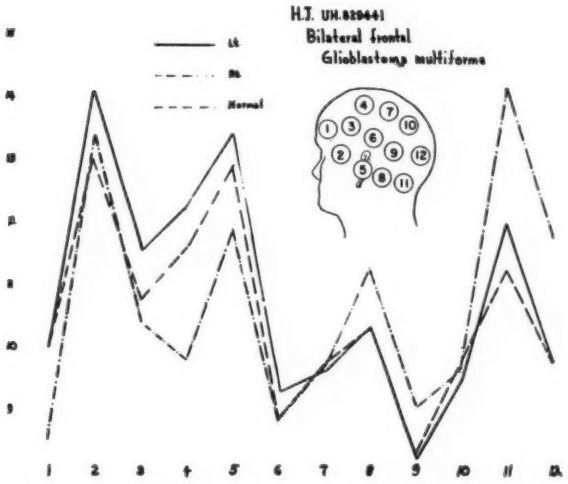


FIG. 9. *Case 3.* Graphic analysis. There is increased radioactivity in the frontal areas on both right and left, with maximum on the left. The patient had a large glioblastoma in the frontal region that crossed the mid line.

the frontal areas on both sides (Fig. 9). Positions 1 are high as compared with the normal, and so are positions 2. The most abnormal are positions 4, which normally should be on the same level as positions 7 and 10. In general, the axis of the graph is tilted upward in the 1st zone, indicating a mid-line lesion in the anterior cranial

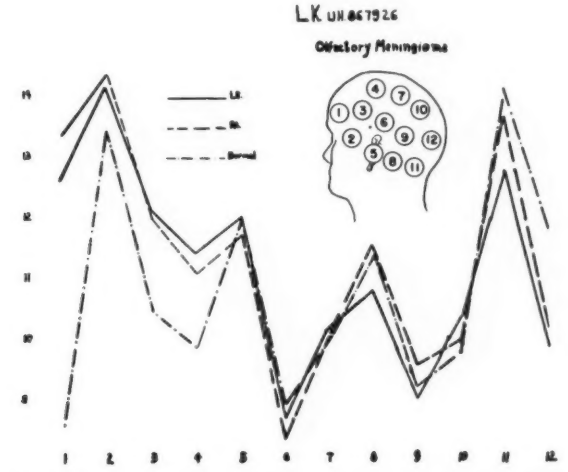


FIG. 10. *Case 4.* Graphic analysis (olfactory groove meningioma). The alteration in counts from the normal is apparent in the frontal region and is present on both right and left, indicating a mid-line or near mid-line lesion.

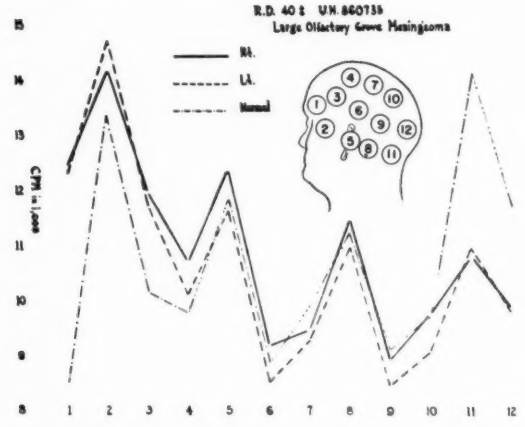


FIG. 11. *Case 5.* Graphic analysis (olfactory groove meningioma). Again there is an increase in counts in the frontal region bilaterally suggestive of a tumor located near the mid line.

fossa. The high amplitude of positions 4 would suggest, in addition, that the lesion is not deep. The 3rd zone in this case, that is the occipital part of the graph, occupies a lower level than the normal. There are two reasons for this manifestation. First, as has been mentioned before, this part of the graph has much more variation in amplitude than the rest of the graph because of anatomical variations of the nuchal soft tissues. Second, the graphs are made with a conversion factor which is dependent upon the relationship of the total counts in the "normal" to that of the "unknown"; the high counts in the frontal regions in this case would necessarily depress the

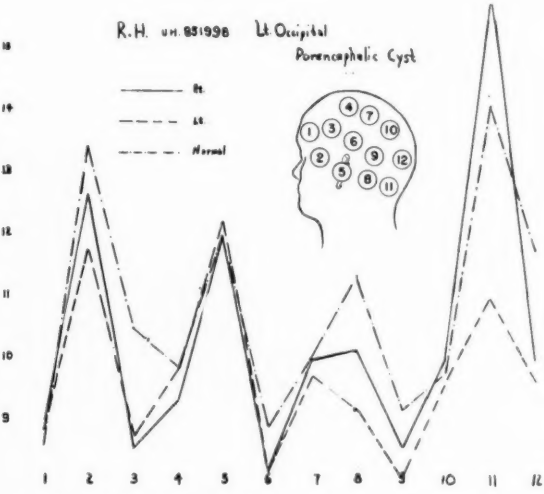


FIG. 12. *Case 6.* Graphic analysis (left occipital porencephalic cyst). See text for explanation of the obvious increase in activity in the occipital area, especially on the right as compared to the left.

amplitude in the posterior positions. Therefore, this apparently significant decrease of amplitude in the 3rd zone of this case is not significant.

Case 4, and Case 5. L.K., UH 867926, and R.B., UH 860735. Meningiomas, olfactory groove. In neither of these patients was the tumor localized by the method of asymmetry because the activity in the tumor contributed equally to the counts on both sides. But on graphic analysis (Figs. 10 and 11) it is very apparent that there is a mid-line lesion in both cases. There is a marked tilt of the 1st zone upward in both cases. Positions 1, 2, 3, and 4 are much higher than those in the normal graph. Here again, positions 4 are higher bilaterally than those of 7 and 10, which, in these cases, is a strong indication of some abnormality being present. Observe that the occipital positions of Case 5 are lower bilaterally than the normal level. Again this is not significant on the basis of reasons previously described in connection with Case 3.

Case 6. R.H., UH 851998. Porencephalic cyst, left occipital lobe. The interpretation by asymmetry was quite equivocal in this case for it showed an increase of activity in the right occipital area. On graphic analysis the abnormality is evident in the 3rd zone (Fig. 12). On the left side, from positions 7 to 12 inclusive, with the exception of position 10, all the activities are somewhat lower than the normal level, with position 11 being the most apparent. The rest of the graph, however, does not show any definite abnormality. It is noted again that positions 4, 7 and 10 are generally isometric, indicating no abnormality over the vertex, yet there is so much deviation from the normal of the occipital positions, especially with asymmetry of positions 11, that a lesion should be suspected. In this case, the porencephalic cyst occupied the whole occipital lobe and it is not at all surprising to find such low activity on the left side.

RESULTS

During the past year 69 patients were given RISA and records were made with a scintillation counter. The data were analyzed by both asymmetry and by the graphic method. Both were analyzed without clinical or other laboratory data. The cases included neoplasms, porencephalic cysts, and acute vascular lesions. The results showed 87 per cent correctly localized by the graphic analysis method and 68 per cent by the usual technique of asymmetry.

CONCLUSION

The graphic method is valuable in localizing mid-line neoplastic lesions with increased radioactivity and peripherally located cystic lesions. These are generally not localizable by the routine technique. Six cases have been presented to demonstrate the use of this technique. The method, of course, is by no means useful in all cases. Small lesions, especially those without associated cerebral edema, i.e., most pituitary tumors, take up too little total tracer to alter the peripheral counts. Posterior fossa tumors, which present a very difficult problem of localization with the usual methods of isotope encephalometry since the normal variation in counting level over the posterior fossa is unpredictable, are, for the same reason, difficult problems with the graphic analysis technique. In these instances definite localization can hardly be made without clinical knowledge of the case under study.

THE EFFECT OF CAROTID ARTERIOGRAPHY, USING 35 PER CENT DIODRAST, ON THE PROTEIN CONTENT OF THE SPINAL FLUID*

GEORGE T. TINDALL, M.D.

Department of Neurosurgery, Duke University School of Medicine and Duke Hospital, Durham, North Carolina

(Received for publication December 22, 1956)

THE effect of carotid arteriography on the protein content of the cerebrospinal fluid seems important to determine since a diagnostic lumbar puncture is often deferred until after arteriography has been performed. Bassett *et al.*[1] noted a significant rise in cerebrospinal fluid protein in experimental animals 30 minutes after the intracarotid injection of 35 per cent Diodrast. The quantity of contrast media employed in these animals, however, was in excess of equivalent amounts used clinically in humans and an elevation in spinal fluid protein might be expected. Stern[4,5] observed no change in protein values of cerebrospinal fluid in 3 patients from 30 minutes to 5 hours after carotid arteriography using relatively large amounts of 35 per cent Diodrast. This series, however, is too small to draw any valid conclusions. Stein and Fink[3] examined the protein content, cellular pattern, and color of spinal fluid from 21 patients prior to and from 12 to 24 hours following cerebral arteriography using 35 per cent Diodrast. They found an elevation in protein content in 1 case, whereas in the remaining 20 patients, the change was insignificant. The present work largely confirms the studies of Stein and Fink but, in addition, shows that a significant decrease in cerebrospinal fluid protein may occur both in patients following arteriography and in controls after a lumbar puncture.

METHOD

A total of 36 patients undergoing carotid arteriography was studied. The contrast substance used in each case was 35 per cent Diodrast in amounts ranging from 20 to 100 cc. All arteriograms were performed by percutaneous puncture of the carotid artery. The various neurologic entities being investigated by arteriography in this group are shown in Table 1.

A lumbar puncture was performed on each patient from 1 to 12 hours before and 24 hours after arteriography. A differential cell count on the spinal fluid was done immediately after the puncture. The fluid was centrifuged and the supernatant material was placed in the deep freeze until the protein determinations were done.

* This investigation has been made with the assistance of a grant from the Committee on Research, Council of Pharmacy and Chemistry, American Medical Association and by a grant from the National Paraplegic Foundation.

TABLE 1

Diagnosis in 36 cases of carotid arteriography with 35 per cent Diodrast

Diagnosis	No. Patients
No intracranial lesion demonstrated	15
Brain tumor	4
Cerebral thrombosis	4
Intracranial aneurysm with subarachnoid hemorrhage	4
Intracranial aneurysm without subarachnoid hemorrhage	3
Epilepsy	3
Intracranial A-V fistula (without subarachnoid hemorrhage)	2
Intracerebral hematoma	1
Total	36

The protein content of cerebrospinal fluid in each case was determined by the biuret method. The reagent used in this procedure was prepared as described by Gornall *et al.*[2] In the determination, 8 cc. of this reagent are added successively to separate Coleman tubes, each containing 2 cc. of spinal fluid; 2 cc. of 0.85 per cent sodium chloride solution, which serves as a blank; and 2 cc. of a standard containing 30 mg. per cent of protein. The resulting solutions were allowed to stand at room temperature for 30 minutes, following which the optical density of each was read in a Coleman Junior spectrophotometer set at a wave length of 540 mμ. The protein content of the spinal fluid could then be obtained from the expression:

$$\text{Mg. \% CSF protein} = \frac{\text{mg. \% protein standard} \times \text{optical density CSF protein}}{\text{optical density of protein standard}}$$

The protein standard was prepared by suitable dilution of a known concentration of bovine albumin (Armour Laboratories) with a solution of 0.85 per cent sodium chloride.

Fourteen patients who did not undergo arteriography served as controls. In this group, 2 lumbar punctures were performed on each patient 24 hours apart and the protein content of the cerebrospinal fluid was determined as described above.

RESULTS

The changes in the protein content of the cerebrospinal fluid 24 hours following carotid arteriography using 35 per cent Diodrast are summarized in Table 2. An increase or decrease of 10 mg. per cent or more is considered to be a significant change. A decrease in the protein content, ranging from 10 to 20 mg. per cent, occurred in 8 patients, whereas in 27 patients there was no significant change from control values. Only 1 patient in this series of 36 cases showed an increase in protein content 24 hours following arteriography. This was a 33-year-old man who had an intracerebral hematoma. The spinal fluid protein in this case was 96 mg. per cent before and 120 mg. per cent 24 hours after arteriography.

TABLE 2

Effect of carotid arteriography, using 35 per cent Diodrast, on protein content of the cerebrospinal fluid in 36 patients

Protein Content of Cerebrospinal Fluid 24 Hours after Arteriography	No. Patients
No change	27
Greater than 10 mg.% increase	1
10–20 mg.% decrease	8
Total	36

There was one adverse reaction following arteriography and it was assumed to be secondary to the procedure. This occurred in a 45-year-old man with a left parietal lobe tumor and consisted of a marked right hemiparesis which was first noted 4 hours after arteriography. It is interesting to note in this patient that the cerebrospinal fluid protein was 80 mg. per cent 1 hour before the arteriogram and 82.5 mg. per cent 24 hours later.

Fourteen patients served as controls for those in whom arteriography was carried out. In the control group, 2 lumbar punctures were performed

TABLE 3

Effect of lumbar puncture on protein content of the cerebrospinal fluid in 14 patients (control group)

Protein Content of Cerebrospinal Fluid 24 Hours after Lumbar Puncture	No. Patients
No change	10
Greater than 10 mg.% increase	0
10–20 mg.% decrease	4
Total	14

on each patient 24 hours apart. The results of this study are shown in Table 3. Ten patients showed no significant change and 4 showed a 10 to 20 mg. per cent decrease in cerebrospinal fluid protein 24 hours following a lumbar puncture.

There were no consistent changes in the cellular pattern of the spinal fluid either in the group undergoing arteriography or in the controls.

COMMENT

The changes in cerebrospinal fluid protein 24 hours following carotid arteriography with 35 per cent Diodrast were not significantly different from those 24 hours following a lumbar puncture. A 10 mg. per cent or greater decrease occurred in 8 out of 36 patients undergoing arteriography and in 4 out of 14 controls. The reason for the decrease is not clear. Sweet *et al.*[6]

have shown that under normal conditions very little mixing of ventricular, cisternal and lumbar fluids occurs. It may be that following a lumbar puncture, leakage of cerebrospinal fluid at the site of the dural puncture allows the ventricular and cisternal fluids to mix more than normally with the lumbar subarachnoid fluid. Since ventricular and cisternal fluids each have a protein content less than that found in the lumbar subarachnoid space, this would result in a lower value 24 hours later.

The failure to demonstrate an elevation in the protein content of the cerebrospinal fluid after arteriography in the patient who experienced a reaction to the procedure is not surprising since Stein and Fink[3] did not observe a significant change in 3 patients who showed a transient hemiparesis following arteriography.

SUMMARY

The protein content of cerebrospinal fluid was determined prior to and 24 hours following carotid arteriography using 35 per cent Diodrast in 36 patients. Of these, 27 showed no significant change; 8 showed a 10 mg. per cent or greater decrease; and 1 showed a 24 mg. per cent increase in the protein content of the cerebrospinal fluid following arteriography.

The changes in protein content of the cerebrospinal fluid in a control group of 14 patients, 24 hours following a lumbar puncture, with 1 exception, were similar to those following arteriography.

There were no consistent changes in the cellular pattern of the cerebrospinal fluid either in the group undergoing arteriography or in the controls.

REFERENCES

1. Bassett, R. C., Rogers, J. S., Cherry, G. R., and Gruzhit, C. The effect of contrast media on the blood-brain-barrier. *J. Neurosurg.*, 1953, *10:* 38–47.
2. Gornall, A. G., Bardawill, C. J., and David, M. M. Determination of serum proteins by means of the biuret reaction. *J. biol. Chem.*, 1949, *177:* 751–766.
3. Stein, J. M., and Fink, M. Spinal fluid findings following cerebral angiography. *Neurology*, 1953, *3:* 137–138.
4. Stern, W. E. The iodine content of the cerebrospinal fluid following Diodrast angiography. *Trans. Amer. neurol. Ass.*, 1953, 266–268.
5. Stern, W. E. Personal communication.
6. Sweet, W. H., Brownell, G. L., Scholl, J. A., Bowsher, D. R., Benda, P., and Stickley, E. E. The formation, flow and absorption of cerebrospinal fluid; newer concepts based on studies with isotopes. *Res. Publ. Ass. nerv. ment. Dis.*, 1956, *34:* 101–159.

PROGNOSIS OF SUBARACHNOID HEMORRHAGE

A COMPARISON BETWEEN PATIENTS WITH VERIFIED ANEURYSMS AND PATIENTS WITH NORMAL ANGIOGRAMS

G. AF BJÖRKESTEN, M.D., AND HENRY TROUPP, M.D.
Department of Neurosurgery, Finnish Red Cross Hospital, Helsinki, Finland

(Received for publication December 23, 1956)

IT IS a well-known fact that the prognosis of subarachnoid hemorrhage is serious; about a third of the patients die from the first bleeding, another third from recurrent hemorrhage, whereas a considerable number of the survivors are incapacitated. [1,6,9,13,14,21] Most authors do not differentiate between the prognosis for patients with proven aneurysms and the prognosis for patients with normal angiographic findings; we have studied these two groups separately. Parkinson[18] has made a similar survey on a smaller scale; out of his 8 conservatively treated patients with verified aneurysm, 7 died of recurrent hemorrhage within 2 to 8 weeks, whereas 21 patients without any verifiable aneurysm were alive after follow-up periods varying from 1 to 5 years. Among 18 conservatively treated patients with aneurysm Hamby[8] had a mortality of 61 per cent. Dunsmore and Polcyn[4] reported 81 cases of subarachnoid hemorrhage without angiographically verified aneurysm. Of these patients, they managed to trace 71, 19 of whom had died from recurrent hemorrhage, all but 1 during the first year after the initial hemorrhage, the mortality rate being 27 per cent. However, bilateral angiography had not been performed on all patients in this series; at autopsy an aneurysm was found in 1 of them.

MATERIAL

We have divided our material into 2 groups.

(1) "*Aneurysm series*": 40 patients with intracranial aneurysm confirmed by angiography or at autopsy, and typical clinical symptoms and signs of subarachnoid hemorrhage even in those cases in which lumbar puncture was not performed during the episode of bleeding.

(2) "*No-aneurysm series*": 61 patients with subarachnoid hemorrhage verified by lumbar puncture, but with normal bilateral carotid angiograms.

The first group comprises all those patients who were admitted to the neurosurgical department of the Finnish Red Cross Hospital from 1938 to 1955, and on whom no operations were performed, either because they were hospitalized during the early period when we did not treat aneurysms surgically, or because they refused to be operated on or operation was deemed inadvisable. The second series includes all patients from the same period that fulfill the criteria stated above. This means that nearly all patients up to 1947 had to be omitted, because of the fact that, until then, bilateral angiography was seldom performed. Only a small number of the patients were submitted to vertebral angiography.

It should also be mentioned that in either series very few of the patients were admitted to the hospital during the acute stage of bleeding, the majority of them having been treated in other hospitals during that time.

During the same period about 100 aneurysms were treated surgically, the majority by intracranial approach.

AGE AND SEX

In the aneurysm series the ages of the patients ranged from 19 to 68 years, the average age being 41. The sex distribution was 20 males and 20 females.

In the no-aneurysm series the ages ranged from 16 to 64 years, the average age being 40. The sex distribution was 27 males and 34 females.

NEUROLOGICAL SIGNS

With regard to possible prognostic significance we noted the occurrence of unconsciousness and/or major neurological signs in association with the attacks of hemorrhage. Hemiplegia or hemiparesis were regarded as major neurological signs, as were convulsions, and severe or long-standing mental symptoms, but not ophthalmoplegia, which can occur without simultaneous bleeding.

In the aneurysm series 10 patients were unconscious and displayed major neurological signs during the attack of bleeding; 4 displayed major neurological signs only, and 10 were unconscious only.

In the no-aneurysm series the ratio was: 6 out of 61 had been unconscious with neurological signs, 14 displayed major neurological signs only, and 19 were unconscious without pareses or mental symptoms.

NUMBER OF ATTACKS BEFORE DIAGNOSIS

In the aneurysm series 28 patients had had only 1 attack prior to definite diagnosis; 11 patients had had 2 attacks. One patient had chronic symptoms (probably several small bleedings).

In the no-aneurysm series 46 patients had had 1 subarachnoid hemorrhage only, 13 had had 2, and 2 patients had had 3 attacks.

Here we have also counted unverified episodes of bleeding, for the verification of a subarachnoid hemorrhage in a patient makes it most likely that recurrent attacks with similar clinical symptoms and signs stem from the same source.

HYPERTENSION

The material was divided into 2 groups: normotensive patients and hypertensive patients. All patients with a blood pressure above 150/100 were grouped as hypertensive.

In the aneurysm series 13 patients were hypertensive and 25 normotensive (in 2 cases no blood pressure was recorded in the file).

In the no-aneurysm series 12 patients were hypertensive and 49 normotensive.

ANATOMICAL LOCATION OF ANEURYSMS, AND MORTALITY RATE

Table 1 shows the location of the aneurysms and the result of the follow-up.

The patient recorded as having multiple aneurysms had one on both middle cerebral arteries respectively, and one on the left pericallosal artery. As it could not be decided clinically which of them had ruptured, we have placed this patient in a separate group.

All deaths have occurred from verified recurrent subarachnoid hemorrhage or at least there have been suggestive clinical signs. One of the patients, here recorded as living, died from thymoma 8 years after his first and only hemorrhage.

Table 1 shows that conservatively treated aneurysms of the internal carotid artery carry the worst prognosis, whereas patients with aneurysms

TABLE 1

Location of aneurysms, and mortality

Location	Number of Patients	Deaths	Living
Internal carotid artery	14	10	4
Middle cerebral artery	11	6	5
Anterior cerebral and anterior communicating arteries	11	3	8
Vertebral system (basilar artery)	3	3	—
Multiple aneurysms	1	—	1
	—	—	—
Total	40	22	18

of the anterior cerebral or anterior communicating artery seem to have the best chance of survival. There were only 3 patients with aneurysm of the vertebral system; all of them died from the first bleeding and the aneurysm was found at autopsy.

In our series, patients with aneurysm of the left hemisphere showed a higher mortality rate than did those with a lesion on the right side. Out of 13 patients with aneurysms of the left internal carotid or the left middle cerebral artery, 11 died; out of 12 patients with aneurysms of the corresponding arteries on the right side 5 died. However, these numbers are too small to be of any statistical significance.

Among the 18 survivors, only 2 had had recurrent subarachnoid hemorrhages during the follow-up period.

MORTALITY IN NO-ANEURYSM SERIES

During the follow-up time 3 patients in the no-aneurysm series died from recurrent hemorrhage. On 1 of them autopsy was performed but no aneurysm was found. In the other 2 cases autopsy was not performed.

Also in this series 2 patients had survived recurrent hemorrhage during the follow-up period. The remaining 56 patients had had no bleedings.

PROGNOSTIC FACTORS

A comparison between the 2 groups shows that in the aneurysm series the mortality rate is more than 10 times as high as in the no-aneurysm series. Consequently the presence or the absence of an angiographically demonstrable aneurysm is the most significant prognostic factor.

Out of the total number of 25 deaths (in both series) 22 occurred within the first year after the initial hemorrhage (Table 2), 15 of these within the first 2 months. The fact that a patient survives the first 12 months after the hemorrhage does not, however, guarantee that the danger is passed; 2 patients in the aneurysm series and 1 in the no-aneurysm series died after this period, one of them (aneurysm) as late as 7 years after the first bleeding.

TABLE 2

Time of survival after initial hemorrhage

Series	Months				Years				
	0–1	1–2	2–6	6–12	1–2	2–3	3–5	5–10	>10
Aneurysm series:									
Deaths	8	5	5	2			1	1	
Living					10	2	3	1	2
No-aneurysm series:									
Deaths	1	1				1			
Living					25	15	10	8	

Dandy[3] has reported still longer intervals. Consequently, the number of deaths may increase in the future.

No correlation between unconsciousness in connection with hemorrhage and chance of survival was found; nor does the prognosis appear to be influenced by the presence of major neurological signs in connection with the bleeding. Consequently, the condition of the patient during the attack of bleeding gives us no clue as to the prognosis.[12] On this point, however, other opinions have been advanced.[10,14] In these series the chances of survival are apparently uninfluenced by hypertension; in the no-aneurysm series none of the 3 patients who died had increased blood pressure, and out of the 13 hypertensive patients in the aneurysm series 7 died and 6 are alive, a ratio that equals the ratio between living and dead in the total aneurysm series.

At the follow-up the patients were asked to describe their working capacity as a result of the bleeding as "normal," "impaired," or "nil"; in some cases the disability was caused by other diseases and these patients have been grouped separately (Fig. 1).

With regard to the aneurysm series it is worth mentioning that 6 patients out of the 7 with normal working capacity were under 40 years of age at the episode of bleeding, whereas 4 patients out of the 5 completely incapacitated were past 40 at the time of bleeding. We should also like to stress that 6 patients out of the 7 with normal working capacity had their aneurysm

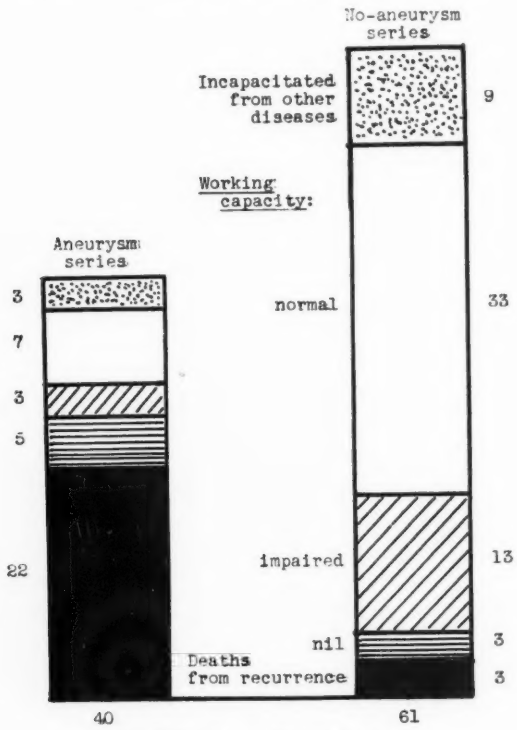


FIG. 1. Present condition of patients.

on the anterior cerebral or the anterior communicating artery. Thus, conservatively treated aneurysms of these arteries seem to carry the best prognosis both as regards chance of survival and working capacity.

In the no-aneurysm series the working capacity of the patients was, on the whole, better than in the aneurysm series. Out of the 58 survivors 33 patients regarded themselves as completely fit for work. Only 3 patients were, according to their own opinion, completely disabled as a result of the hemorrhage. The majority of the 13 patients with impaired working capacity had only slight and vague symptoms, as for instance, headache, dizziness, and tiredness, which, in fact, need not always have been caused by the bleeding. No influence of age can be proved, but it ought to be pointed out that 10 out of the 12 hypertensive patients belonged to the partly or entirely incapacitated groups; in fact, symptoms of this kind may equally well be caused by increased blood pressure.

DISCUSSION

The question that arises on comparing the 2 groups mentioned above is: how certain can we be that patients with normal carotid angiograms have

no aneurysm? Even though angiographic technique has been greatly improved, this does not guarantee that every aneurysm is visualized. In the Finnish Red Cross Hospital for the last 3 years we have done the angiograms in three projections: lateral, anteroposterior, and oblique. The oblique view has proved to be specially important in the visualization of aneurysms of the anterior communicating artery. Up to 1953 only lateral and anteroposterior projections were used. In 1 case bilateral angiography, with all three views, showed no aneurysm; when, after some time, the patient died from recurrent hemorrhage, autopsy revealed an aneurysm of the anterior communicating artery. This shows that an aneurysm can be overlooked despite thorough examination.

Opinions as to the value of stereoscopic angiograms still differ; this technique has not been used at the Finnish Red Cross Hospital.

One shortcoming in our series is that vertebral angiography was performed on only a few of our patients. It is, however, highly probable that very few aneurysms have been overlooked for this reason; aneurysm of the vertebral area and especially on the basilar artery is very seldom found in patients who have survived attacks of subarachnoid hemorrhage[5,11,20] because most of them succumb to the attacks. Peripherally located aneurysms of the vertebral system can be visualized if the posterior cerebral artery is visible on the carotid angiogram; in that case vertebral angiography is of little value.[20] In our series the posterior cerebral artery could be visualized by carotid angiography in somewhat less than one-fourth of the cases, but no aneurysm was found in this region. Yet, an arteriovenous malformation, filling from the vertebral artery only, must be taken into account; in such cases the hemorrhage is mainly venous and the patients' chances of survival are much greater. We have observed 1 such case; the patient has survived several bleedings.

The source of bleeding in cases of subarachnoid hemorrhage with normal angiograms, still presents an unsolved problem. The possibility of an overlooked aneurysm has been mentioned above. The fact that among our cases of subarachnoid bleeding the incidence of angiographically verified aneurysms is lower than in Löfstedt's series,[11] goes to prove that we have not been able to visualize all aneurysms. The circumstance that 2 out of the 3 patients who died in our no-aneurysm series succumbed within 2 months after the initial hemorrhage also speaks strongly in favour of an overlooked aneurysm, this period being the most critical for patients with aneurysms that have bled.[18,21] On the other hand we should like to point out that in our no-aneurysm series no arterial vasospasm worth mentioning could be seen. Such a vasospasm is fairly common in patients with aneurysm, especially during the first weeks after the bleeding.[16,17] Yet the absence of vasospasm may have been ascribable to the comparatively late stage at which the angiograms were done.

The possibility of a spontaneous thrombosis of a small ruptured aneurysm should perhaps also be taken into account.[7,12] Spontaneous thrombosis of

larger aneurysms is unlikely; we have never seen spontaneous thrombosis of an angiographically verified aneurysm, whereas an enlargement of the aneurysmal sac is a common finding after a bleeding. It is also a well-known fact that even a partial clipping of the aneurysmal stalk does not always cause thrombosis, although the application of the clip causes a lesion of the intima which ought to increase the tendency to clot.

In 1951 Margolis *et al.*[15] described small angiomatous malformations causing intracerebral hematomas. One of their 4 patients had blood in the cerebrospinal fluid. These malformations may be a significant etiological factor of subarachnoid hemorrhage, being so small that they are not visible in angiograms. Telangiectases as a cause of subarachnoid bleeding have also been described.[2,19]

In spite of all imaginable methods of examination, all clinical and pathological series of patients with subarachnoid hemorrhage contain cases in which the cause of the bleeding cannot be traced. Whatever the etiology, the fact remains that a patient with a normal angiogram has a good chance of survival whereas patients with aneurysms not treated surgically show a very high rate of mortality.

SUMMARY AND CONCLUSIONS

A series of 40 patients with intracranial arterial aneurysm verified by angiography or at autopsy, and another series of 61 patients with verified subarachnoid hemorrhage but normal bilateral carotid angiograms, are reported. All patients were treated conservatively. In the former group the mortality from recurrent hemorrhage was 55 per cent (22/40), in the latter, 5 per cent (3/61). Aneurysms of the internal carotid artery had the worst prognosis; those of the anterior cerebral and the anterior communicating arteries had the best. At the follow-up the patients in the no-aneurysm series showed a better condition and a better working capacity than did the patients in the aneurysm series.

The possible etiology of the hemorrhage in the patients with normal carotid angiograms is discussed.

It is not relevant to speak of the prognosis of subarachnoid hemorrhage in general; it is essential to differentiate between cases of aneurysms and cases in which the angiographic findings are normal. Even if the source of bleeding in the no-aneurysm group is unknown, a normal bilateral carotid angiogram points to a better prognosis than when an aneurysm is found, but left unoperated upon.

ADDENDUM

After finishing this paper we were informed that autopsy had been performed on another of the 3 deceased patients in the "no-aneurysm" group. A ruptured aneurysm of the right posterior cerebral artery was found.

REFERENCES

1. Ask-Upmark, E., and Ingvar, D. A follow-up examination of 138 cases of subarachnoid hemorrhage. *Acta med. scand.*, 1950, *138:* 15–31.
2. Christensen, E. Pathologie der intrakraniellen Blutungen. In: *Handbuch der Neurochirurgie.* H. Olivecrona and W. Tönnis, Ed. Berlin: J. Springer, 1956, *3:* 703–736.
3. Dandy, W. E. Intracranial arterial aneurysms. *Ithaca, N. Y.: Comstock Publishing Co., Inc.*, 1944, viii, 147 pp.
4. Dunsmore, R. H., and Polcyn, J. L. Subarachnoid hemorrhage: prognostic factors. *J. Neurosurg.*, 1956, *13:* 165–169.
5. Falconer, M. A. Surgical pathology of spontaneous intracranial haemorrhage due to aneurysms and arteriovenous malformations. *Proc. roy. Soc. Med.*, 1954, *47:* 693–700.
6. Hamby, W. B. Intracranial aneurysms. *Springfield, Ill.: Charles C Thomas*, 1952, xxi, 564 pp.
7. Hamby, W. B. The aneurysmal origin of nonfatal subarachnoid hemorrhage. An angiographic survey of 53 cases. *J. Neurosurg.*, 1953, *10:* 35–37.
8. Hamby, W. B. The surgical treatment of intracranial aneurysms. Results in angiographically located lesions. *Arch. Neurol. Psychiat., Chicago*, 1956, *75:* 345–349.
9. Hyland, H. H. Prognosis in spontaneous subarachnoid hemorrhage. *Arch. Neurol. Psychiat., Chicago*, 1950, *63:* 61–78.
10. Jacobson, S. A. Analysis of some factors in spontaneous subarachnoid hemorrhage. *Arch. Neurol. Psychiat., Chicago*, 1954, *72:* 712–719.
11. Löfstedt, S. Subarachnoidalblödningarnas etiologi—en klinisk-röntgenologisk studie. *Nord. med.*, 1952, *47:* 327–328.
12. Logue, V. Surgery in spontaneous subarachnoid haemorrhage. Operative treatment of aneurysms on the anterior cerebral and anterior communicating artery. *Brit. med. J.*, 1956, *1:* 473–479.
13. Magee, C. G. Spontaneous subarachnoid haemorrhage. A review of 150 cases. *Lancet*, 1943, *2:* 497–500.
14. Magladery, J. W. On subarachnoid bleeding—an appraisal of treatment. *J. Neurosurg.*, 1955, *12:* 437–449.
15. Margolis, G., Odom, G. L., Woodhall, B., and Bloor, B. M. The role of small angiomatous malformations in the production of intracerebral hematomas. *J. Neurosurg.*, 1951, *8:* 564–575.
16. Norlén, G., and Barnum, A. S. Surgical treatment of aneurysms of the anterior communicating artery. *J. Neurosurg.*, 1953, *10:* 634–650.
17. Norlén, G., and Olivecrona, H. The treatment of aneurysms of the circle of Willis. *J. Neurosurg.*, 1953, *10:* 404–415.
18. Parkinson, D. The problem of spontaneous subarachnoid hemorrhage with proven aneurysms. *J. Neurosurg.*, 1955, *12:* 565–569.
19. Russell, D. S. The pathology of spontaneous intracranial haemorrhage. *Proc. roy. Soc. Med.*, 1954, *47:* 689–693.
20. Sjögren, S. E., Stockholm, Sweden. Personal communication, 1956.
21. Walton, J. N. The prognosis and management of subarachnoid haemorrhage. *Canad. med. Ass. J.*, 1955, *72:* 165–175.

*SPECIAL ARTICLE**

THE CLINICAL USE OF ELECTROENCEPHALOGRAPHY

PAUL C. BUCY, M.D.†

ALTHOUGH there had been earlier suggestions that cerebral activity is associated with electrical alterations, the first reliable demonstration that alterations in electrical potential can be recorded from the brain was published by Hans Berger[1] in 1929. With his many publications in the early 1930's interest began to be shown in this phenomenon. In 1935 Gibbs[6] published (in collaboration with Davis and Lennox) his first paper on the electroencephalogram in epilepsy, and the next year Jasper[9] followed. In 1935 Foerster and Altenburger[5] noted the changes in the electroencephalogram associated with intracranial tumors. In 1936 Walter,[13] and in 1938 Case and Bucy[3] confirmed these observations. Since then the interest in electroencephalography has steadily increased. Unfortunately there has been, at times, an overly enthusiastic clinical utilization of electroencephalography, and claims have been made as to its diagnostic values that are not in accord with scientific observations. It is important, therefore, that we make a dispassionate analysis of electroencephalography. What it is; what uses can be made of it; and, how it can be of assistance in clinical medicine, particularly in clinical neurology and neurological surgery.

By applying a number of electrodes to various parts of the human head, connecting them with suitable amplifiers and recording equipment, it is possible to record alterations in electrical potential between two different points. Commonly these electrodes are applied to the scalp overlying the cerebrum (although at times electrodes may be placed over the cerebellum, in the nasopharynx, on the ears or on the nose). The electrodes are usually flat, round metal discs about 1 cm. in diameter which are attached to the shaved scalp with a suitable adhesive. (Occasionally needle electrodes which can be inserted into the scalp have been used but in general these have been unsatisfactory.) By means of these electrodes variations in electrical potential between any two points can be recorded. Sometimes both points overlie one cerebral hemisphere; sometimes one is on one side and one on the other, and on other occasions comparisons may be made between a point over the cerebrum and a point on an ear or the nose, which the electroencephalographer hopes is "inactive" or "indifferent." It has been shown that the alterations in electrical potential thus recorded may result from changes in electrical potential within the cerebrum, particularly in the cerebral cortex. In fact it is in the hope of studying such changes within the cerebrum that

* Prepared at the request of the Editorial Board of the *Journal of Neurosurgery.*

† Professor of Surgery, Northwestern University Medical School, and Head of the Section on Neurological Surgery, The Chicago Wesley Memorial Hospital, Chicago, Illinois.

the electroencephalogram is made. It is important, however, that the electroencephalographer be thoroughly familiar with all of the other sources that may produce changes in electrical potential between any two of his electrodes, and that may prove misleading in his interpretation. Among the more obvious of these are the action of the heart, showing an electrocardiogram in the record; muscular activity of the fronto-occipitalis muscle or the muscles of the eyes or of mastication; or movement of an electrode in relation to the scalp; or such extraneous factors as electrical defects in the equipment, or the standard 60 cycle electrical current, or the electrical activity of unrelated electrical equipment in or near the hospital or laboratory. It is also important that the electroencephalographer recognize that the electroencephalographic equipment which is now quite standardized is providing him with an incomplete picture of the alterations in electrical potential occurring within the brain. This is true for two principal reasons—the equipment itself and the nature of the examination. The equipment has certain limitations built in it which restrict the frequency of alterations in potential that it is able to record. It is obvious that an ink-writing instrument writing on paper moving at the rate of 3 cm. per second (which is standard) cannot record the high frequencies which are present and which can be recorded, for instance, by a cathode ray oscillograph. The fact that no one knows the significance of these higher frequencies which the present equipment completely ignores is no evidence that these frequencies are less significant than those with which we are more familiar. But more important still is the nature of the entire examination. Alterations in electrical potential in the scalp are being recorded which it is hoped and believed are a reflection of the electrical activity within the cerebrum. At the very outset it must be obvious that recording from 16, 32 or 100 electrodes on the scalp can give but a very rough idea of the electrical activity within the millions of neurons and neuronal connections within the brain itself. In attempting to record the activity of the millions of units within the brain with a mere handful of recording electrodes we must be recording a summation of a large number of intracerebral units. If the intracerebral units were firing completely indiscriminately the alterations in electrical potential of half of them would cancel out the alterations in the other half and the electroencephalogram would be a perfectly flat line, i.e. we would record nothing. But, this complete silence of the electroencephalogram would not mean complete inactivity in the brain. In fact it might be consonant with intense activity. The fact that in the electroencephalogram we record something is evidence that at different times variable numbers of intracerebral units function synchronously, producing a sufficient preponderance of alteration of electrical potential in one direction to be recorded. In fact nervous activity within the brain is commonly associated with asynchronous neuronal activity, while inactivity permits a large number of neurons to assume a synchronous basic or inherent rhythm. Thus when the subject is lying relaxed with his eyes closed the basic alpha rhythm of 8 to 10 waves per second becomes promi-

nent, but when the subject opens his eyes and looks about him, or even thinks of looking at objects while his eyes are still closed, the asynchronous and independent actions of the various neurons interfere with each other, the regular alpha rhythm disappears, and the electroencephalographic waves become irregular and their amplitude decreases. Similarly during sleep the electroencephalogram is characterized by slow waves of high amplitude and runs of very regular more rapid waves called "sleep spindles," both of which are characteristic of synchronous activity of large numbers of cells. Or when the normal activity of areas of the brain is interfered with by pressure from a tumor, or by the edema and inflammation of an encephalitis, or by the relative ischemia of the acute stage of a cerebral thrombosis the electroencephalogram from that area is characterized by slow waves of high amplitude. (Cells that are rendered completely inactive by these or any other processes, of course, have no electrical activity and give rise to no waves.) Likewise, when any disease process seizes control of large areas of the cerebrum, thrusts aside all normal activity and occupies all units with a single activity, as with an epileptic attack, we can see the rapid, high voltage spikes of a grand mal seizure, or the slower but also regular wave and spike discharge of a petit mal attack.

The usable facts of electroencephalography are all empirically derived. It is capable at best of recording only the effects of gross lesions. The statement which has been made that it is the best available measurement of the physiological activity of the brain is completely without foundation. In this same connection it must be borne in mind that the electroencephalogram is incapable of demonstrating the absence or destruction of brain tissue. The electroencephalogram cannot disclose the absence of a lobe of the brain that has been removed surgically. It cannot demonstrate the functional inactivity of a frontal lobe that has been largely separated from the rest of the brain by a lobotomy. Likewise, a quiescent cystic cavity that is not under increased pressure and that is not compressing or irritating the surrounding brain will give no evidence of its presence in the electroencephalogram. An area of brain rendered completely inactive by infarction gives no evidence of its presence in the electroencephalogram. In the acute stage an area of infarction produced by cerebral embolism or thrombosis will so affect the neighboring brain as to give rise to a striking slow-wave focus; but, with the passage of time this slow-wave focus will disappear and the electroencephalogram will again become "normal." However, the "normal" electroencephalogram does not mean that the underlying brain has returned to normal, that the impairment of circulation was only temporary. Not at all. The return to "normal" of the electroencephalogram is most likely to mean that the edema and inflammatory reaction have subsided, while the ischemic brain has died, giving way to scar or cyst formation. (In this connection it should also be noted that Kempinsky[11] has shown in the experimental animal that in the acute stage infarction gives rise to a demarcation potential in the white matter between the infarcted tissue containing dying

segments of axons separated from their cells of origin and the segments contained in the intact area on the other side of the boundary.)

"Normal" electroencephalograms may be misleading in many other ways. They may be unrevealing in the presence of a cerebral neoplasm, notably parasagittal meningiomas.[4] O'Leary[12] stated that infiltrating gliomas of the cerebrum are often difficult to recognize with the electroencephalogram. It has long been recognized that tumors of midline structures, of the third ventricle or in the posterior fossa commonly give rise to a misleading electroencephalographic picture.[4] A "normal" electroencephalogram is obtained in some 10 to 20 per cent of all cases of epilepsy[10] and in approximately one-third of all adult epileptics. Subdural hematomas may be associated with a definitely abnormal electroencephalogram but the tracing is often entirely normal.

On the other hand, the electroencephalogram may be misleading in a positive as well as a negative way. About 10 per cent of individuals with no evidence of any kind of neurological abnormality have abnormal electroencephalograms.[7,10] Usually these "abnormalities" are diffuse and of the type generally encountered in epileptics, rather than localized disturbances such as may be seen with tumors. However, even with tumors and other localized lesions misleading false localizing signs may be encountered. Thus a tumor of the right frontal lobe has been associated with an electroencephalogram that disclosed "abnormalities" localized to the opposite temporal lobe *only*. Jasper and Daly[10] stated that "slow waves in the contralateral frontal lobe have been observed in some cases of neoplasm or abscess of the posterior fossa." It must also be borne in mind that although a localized "slow-wave-focus" is typical of an intracranial neoplasm, the same type of electroencephalographic abnormality may occur with a localized infarct or hemorrhage, a traumatized area, an abscess or a localized area of encephalitis. Hoefer *et al.*[8] reported that in a study of 416 tumors of the cerebral hemispheres (i.e. excluding tumors of the diencephalon, midbrain, brain stem and cerebellum which are notoriously difficult to localize by electroencephalography) only 64 per cent were correctly localized. In another 9 per cent the side on which the tumor was located was correctly indicated. In 21.1 per cent there was no evidence of the presence of the tumor and in 5.3 per cent the localization was erroneous. In discussion of this paper Aird of California and Schwab of Boston reported that their experiences were similar.

Still another matter is of the greatest importance so far as the clinical use of electroencephalography is concerned. Relatively few clinicians rely upon their own interpretation of the tracings obtained from their patients. The study and interpretation of the electroencephalograms is commonly delegated to a specialist in that field. How reliable are such interpretations—how closely do several electroencephalographers agree with each other? Blum[2] has investigated that question. Standard electroencephalographic tracings were obtained from "ten routinely referred patients." Eight of these

were subsequently shown to be suffering from disorders involving the central nervous system. The records were examined by five men trained in electroencephalography and with experience with the interpretation of electroencephalograms varying from over 10 years in three to over 3 years in the least experienced. The percentage of complete agreement on pathology was 40, on localization was 30 and on both combined was only 10. I personally have had experience with a case in which four electroencephalograms were made in three different laboratories. Each tracing was interpreted differently. One was said to indicate a lesion in one frontal lobe, one a lesion in the opposite temporal lobe, one was said to show a generalized dysrhythmia and one was said to be normal. It should be added that repeated neurological examinations disclosed no evidence of any neurological disease and the history was not suggestive of any greater disturbance than a man seeking compensation for a trivial injury.

It is obvious that interpretation of the electroencephalogram is by no means easy and that there is a wide disagreement as to the significance of the tracings obtained among experienced electroencephalographers.

Although electroencephalography has the inadequacies that have been pointed out above, these can in the main be understood and proper allowance made for them. No method of examining patients is perfect but all, including electroencephalography, are useful and have their proper place. Far more serious than the limitations of the method is the misapplication of electroencephalography for purposes for which it should never be used. It is obvious that electroencephalography is but another laboratory procedure with many limitations. It is a highly specialized examination of a patient for a very short period of time—commonly not more than an hour. It can never be a complete examination in itself. It can never take the place of a careful history, a complete neurological and general physical examination, and an evaluation of all of the evidence available including roentgenograms, examinations of the blood and spinal fluid and special tests when indicated. It is but a small part of the examination of the patient. To diagnose or worse to prescribe for the patient on the basis of the electroencephalogram alone; or to testify in court regarding a question of injury to the brain on the basis of the electroencephalogram without having seen and examined the patient, without knowledge of the complete history is inexcusable. To attempt to classify the histological type of tumor present on the basis of the electroencephalogram alone is also inexcusable as well as impossible but it has been done and is indicative of the lack of critical judgment of some electroencephalographers. Such activities can only bring electroencephalography into disrepute.

Electroencephalography has a proper place in neurology and in medicine. It is not a substitute for any other form of examination. It should not be misapplied or misused. It is of little value in psychiatry, or with functional disorders, except in differential diagnosis. It will not help in the recognition of the "functional" psychoses, of mental retardation, psychoneurosis or

migraine, but it may be of great assistance in identifying some of the brain tumors, encephalitic processes and subdural hematomas that masquerade as functional diseases.

REFERENCES

1. Berger, H. Über das Elektrenkephalogramm des Menschen. *Arch. Psychiat. Nervenkr.*, 1929, *87:* 527–570.
2. Blum, R. H. A note on the reliability of electroencephalographic judgments. *Neurology*, 1954, *4:* 143–146.
3. Case, T. J., and Bucy, P. C. Localization of cerebral lesions by electro-encephalography. *J. Neurophysiol.*, 1938, *1:* 245–261.
4. Cobb, W. A. The electro-encephalographic localization of intra-cranial neoplasms. *J. Neurol. Neurosurg. Psychiat.*, 1944, n.s. 7: 96–102.
5. Foerster, O., and Altenburger, H. Elektrobiologische Vorgänge an der menschlichen Hirnrinde. *Dtsch. Z. Nervenheilk.*, 1935, *135:* 277–286.
6. Gibbs, F. A., Davis, H., and Lennox, W. G. The electro-encephalogram in epilepsy and in conditions of impaired consciousness. *Arch. Neurol. Psychiat., Chicago*, 1935, *34:* 1133–1148.
7. Gibbs, F. A., and Gibbs, E. L. Atlas of electroencephalography. *Cambridge, Mass.: L. A. Cummings Co.*, 1941, 221 pp.
8. Hoefer, P. F. A., Schlesinger, E. B., and Pennes, H. H. Clinical and electroencephalographic findings in a large series of verified brain tumors. *Trans. Amer. neurol. Ass.*, 1946, 52–55.
9. Jasper, H. H. Localized analyses of the function of the human brain by the electroencephalogram. *Arch. Neurol. Psychiat., Chicago*, 1936, *36:* 1131–1133.
10. Jasper, H. H., and Daly, D. D. Diagnostic methods: II. Electroencephalography and electrophysiologic methods. In: *Clinical neurology.* A. B. Baker, Ed. New York, P. B. Hoeber, Inc., 1955, *1:* 201–228.
11. Kempinsky, W. H. Steady potential gradients in experimental cerebral vascular occlusion. *Electroenceph. clin. Neurophysiol.*, 1954, *6:* 375–388.
12. O'Leary, J. L. Changing status of electroencephalography in neurologic practice. *Neurology*, 1955, *5:* 827–846.
13. Walter, W. G. The location of cerebral tumours by electro-encephalography. *Lancet*, 1936, *2:* 305–308.

CASE REPORTS AND TECHNICAL NOTES

BILATERAL SYMMETRICAL ANEURYSMS OF INTERNAL CAROTID ARTERY WITHIN THE CAVERNOUS SINUS

CASE REPORT

JACOB SELTZER, M.D.,* AND EVERETT F. HURTEAU, M.D.
Akron City Hospital, Akron, Ohio

(Received for publication September 24, 1956)

Bilateral aneurysms of the internal carotid artery within the cavernous sinus of nontraumatic origin are a rarity, judging by the very few cases of such lesions reported in the literature. Since Sir Gilbert Blane's[2] report of a case in the year 1800, to our knowledge only 10 cases have been described. In only 2 of these 10 cases were bilateral aneurysms suspected and diagnosed ante mortem. In 1[7] the diagnosis was made by angiography, but in view of the patient's advanced age, surgery was not performed; the diagnosis was proven later at autopsy. In the second case,[6] the diagnosis was made by roentgen ray, which showed bilateral parasellar calcifications, but it was never proven. In 2 cases the bilateral aneurysms were incidental autopsy findings.[3,6] Unilateral aneurysms were diagnosed clinically in 4 cases[1,4–6] in which later, at autopsy, bilateral aneurysms were found. In one case[1] there was a cavernous sinus fistula on one side, which may have been traumatic, and an aneurysm on the other side; these were proven by angiography. In another case, which appeared in the French literature in 1949,[8] aneurysms within the cavernous sinus were found at autopsy, but being unable to obtain the journal, we do not know whether the diagnosis was suspected or proven ante mortem.

In the case presented herein, the diagnosis was proven by angiography. It is reported for its interest and its statistical value.

CASE REPORT

M.H., a 47-year-old white female, was first seen in the outpatient medical clinic of Akron City Hospital on Nov. 2, 1955, with a complaint of sudden onset of left supra- and periorbital headache about 3 weeks previously. The headache, which had lasted 3 days and disappeared as suddenly as it had come, was not altered by salicylates. Shortly after the headache appeared she noted a medial deviation of her left eye. There was no history of trauma. The past medical history was noncontributory.

Examination revealed a blood pressure of 200/120 and weakness of the left 6th nerve. There was no exophthalmos and a bruit was not audible. The fundi were normal. Visual acuity was 20/40 in the right eye; vision in the left eye was markedly blurred. Roentgenograms of the skull were normal. Blood count and urinalysis were normal except for a 2-plus urine albumin. Blood test for venereal disease was positive up to 1:32 dilutions. A course of penicillin therapy was instituted because of the positive serology.

Examination on Feb. 8, 1956 revealed left supraorbital and corneal hypesthesia, ptosis of the left lid, dilatation of the left pupil, and complete left extraocular palsy. V.O.S. was 20/30. She complained of throbbing left frontal headache. At no time did she show any signs referable to involvement of structures within the right cavernous sinus.

* Present address: 6952 Sherwood Road, Philadelphia 31, Pennsylvania.

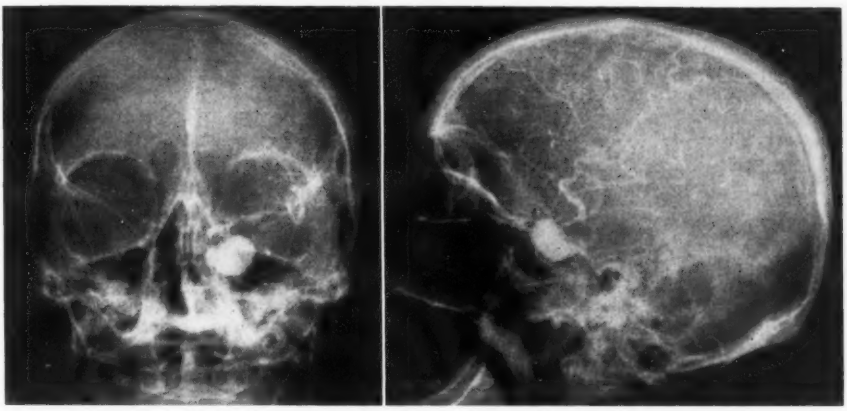


FIG. 1. Left carotid arteriogram demonstrating aneurysm of the left internal carotid artery within the cavernous sinus.

Admission to Akron City Hospital, Feb. 8, 1956. A lumbar puncture was performed; the pressure, appearance, hydrodynamics and laboratory findings were all normal. Orbital roentgenograms were normal. Continuous compression of the left carotid artery for 10 minutes resulted in a slight improvement in the ptosis for the period of the arterial compression.

On Feb. 13, 1956, using 35 per cent Diodrast, a left carotid arteriogram showed a large aneurysm in the cavernous sinus portion of the left internal carotid artery (Fig. 1).

On Feb. 15, 1956, using 35 per cent Diodrast, right carotid arteriography with simultaneous compression of the left carotid artery to check collateral circulation was done. This showed an aneurysm of the right internal carotid artery similar in size and position to that on the left. Satisfactory collateral circulation was demonstrated (Fig. 2).

1st Operation. On Feb. 17, 1956, under local 1 per cent procaine anesthesia, a tantalum clip was placed around the left internal carotid artery in the neck. During the dissection of the structures of the carotid sheath it was necessary to administer some Pentothal sodium intravenously. The clamp was closed completely. The patient's response to pinpoint stimula-

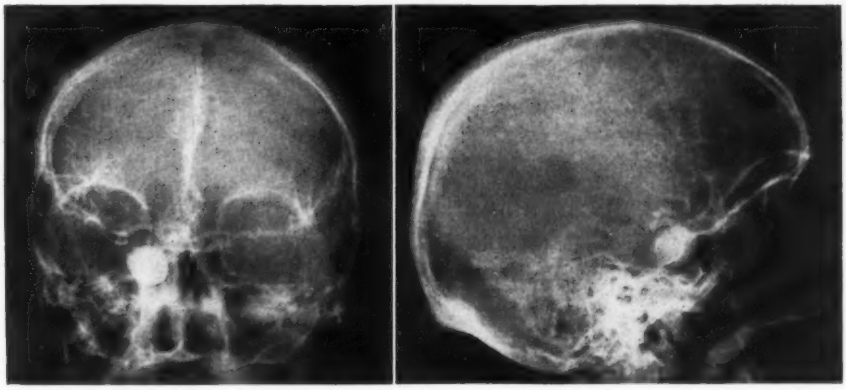


FIG. 2. Right carotid arteriogram demonstrating aneurysm of the right internal carotid artery within the cavernous sinus.

tion was diminished on the right. Eight minutes later the clip was opened to about three-fourths of the normal caliber of the vessel. Good motor response to pinpoint stimulation on the right appeared soon afterwards.

Course. The patient did well postoperatively.

2nd Operation. On Feb. 21, 1956, through the same cervical wound, the tantalum clip was removed and a Selverstone clamp (which had arrived only the previous day) was inserted in its place. The clamp was left entirely open. Beginning on February 22, gradual occlusion of the left internal carotid artery was started; it was completely closed 4 days later without any evidence of aphasia or hemiplegia.

Course. On Feb. 26, 1956, some improvement in the weakness of the left 6th nerve, return of the corneal reflex, and disappearance of the supraorbital hypesthesia were noted. The dilatation of the left pupil diminished and its reaction to light was less sluggish. Complete paralysis of the internal rectus and the ptosis remained unchanged.

There was no further improvement by Mar. 2, 1956 and so it was decided to occlude the left carotid artery intracranially distal to the aneurysm.

3rd Operation. On Mar. 3, 1956 the proposed procedure was performed via a left temporofrontal flap.

Course. The patient did well until approximately 25 hours postoperatively when there developed aphasia, lethargy, and a right hemiplegia which was complete in the upper extremity and partial in the lower extremity. A lumbar puncture yielded blood-tinged spinal fluid under normal pressure. Papaverine was started. It was thought that there might be a propagating thrombus involving the arterial supply of the left cerebral hemisphere.

Within 7 hours active motion of the right lower extremity improved, with return of some of the gross motions of the right upper extremity and some improvement in the aphasia. The hemiplegia and aphasia continued to show slow but steady improvement. On the 2nd postoperative day, another lumbar puncture was performed and it again showed blood-tinged spinal fluid under normal pressure.

Within the next hour, even before anticoagulant therapy was instituted, there was great improvement in the aphasia and hemiplegia. Complete return of function of the right lower extremity and disappearance of the aphasia occurred within the next 24 hours. The weakness of the right upper extremity showed steady improvement aided by physical therapy.

At the time of her discharge from the hospital on Mar. 19, 1956, there remained only a very slight weakness in the right upper extremity. However, there was no improvement at all in the palsy of the left 3rd nerve.

She was last seen on Aug. 24, 1956. At this time she showed no ptosis, no weakness of the external ocular muscles, no pupillary asymmetry and no sensory change referable to the trigeminal nerve. Motor function and superficial and deep reflexes were normal in the right lower extremity. There was very mild limitation of fine movements of the right hand. She had no aphasia. There were no findings attributable to the aneurysm of her right carotid artery.

SUMMARY

A case of bilateral aneurysm of the carotid arteries within the cavernous sinuses is presented. The symmetry of these lesions is striking. Only the one on the left caused symptoms and was treated definitively. The importance of bilateral carotid arteriography even in apparently simple diagnostic problems is illustrated.

The authors are perplexed about therapy of the aneurysm on the right should it become symptomatic, and would be most grateful for pertinent suggestions.

REFERENCES

1. Alpers, B. J., Schlezinger, N. S., and Tassman, I. M. Bilateral internal carotid aneurysm involving cavernous sinus. Right carotid artery-cavernous sinus fistula and left saccular aneurysm. *Arch. Ophthal., N. Y.*, 1951, *46:* 403–407.

2. Blane, G. Cited by Hamby.[5]
3. Dial, D. L., and Maurer, G. B. Intracranial aneurysms. Report of 13 cases. *Amer. J. Surg.*, 1937, n.s. *35:* 2–21 (see Case XIII).
4. Hamby, W. B. Intracranial aneurysms of the internal carotid artery and its branches. *J. int. Coll.* Surg., 1942, *5:* 216–222.
5. Hamby, W. B. Intracranial aneurysms. *Springfield, Ill.: Charles C Thomas*, 1952, xxi, 564 pp. (see p. 107, Case 1).
6. Jefferson, G. On the saccular aneurysms of the internal carotid artery in the cavernous sinus. *Brit. J. Surg.*, 1938, *26:* 267–302.
7. Logue, V. Saccular aneurysms of the internal carotid artery in the cavernous sinus occurring bilaterally. *Brit. J. Surg.*, 1951, *39:* 181–182.
8. Mahoudeau, D., Daum, S., George, and Rosier. Ophtalmoplégie double révélatrice d'un anévrysme intracranien bilatéral de la carotide interne. Intérêt diagnostique de l'artériographie. *Bull. Soc. méd. Hop., Paris*, 1949, *65:* 503–509. Cited by Alpers *et al.*[1]

PARTIAL GLOSSOPHARYNGEAL NEURALGIA ASSOCIATED WITH SYNCOPE

HENDRIK J. SVIEN, M.D., NORMAN C. HILL, M.D., AND DAVID D. DALY, M.D.
Section of Neurologic Surgery and Section of Neurology, Mayo Clinic and Mayo Foundation, Rochester, Minnesota*

(Received for publication November 7, 1956)

The syndrome of typical glossopharyngeal neuralgia is well known. Weisenburg,[20] in 1910, reported a case in which pharyngeal pain was not relieved by partial section of the gasserian ganglion. Sicard and Robineau,[17] in 1920, reported 3 more cases in which a condition that probably was glossopharyngeal neuralgia was treated surgically. The operations were not entirely parallel but they can be considered to have been extensive as regards the 9th and 10th cranial nerves and also as regards a sympathetic structure that probably was the superior cervical ganglion. In 1 of the 3 cases the 12th cranial nerve was cut. Harris,[5] in 1921, described the syndrome and attributed it to neuralgia of the glossopharyngeal nerve. In 1924, Adson[1] treated 2 patients with glossopharyngeal neuralgia by extracranial section of the 9th cranial nerve and suggested an intracranial approach to that nerve, which, according to Love,[8] was employed in 1925. However, the first reported successful intracranial section of the 9th nerve was performed by Dandy[2] in 1927; it was then possible to chart accurately the sensory distribution of the glossopharyngeal nerve.

Pain restricted to the ear is rare in glossopharyngeal neuralgia; only 2 undoubted cases have been reported. In a case described by Reichert[13] in 1934, the pain occurred "deep in the ear" and "in and about the external auditory meatus." The pain could be reproduced by intracranial direct stimulation of the 9th nerve. A pharyngeal trigger point was identified. The pain was relieved by intracranial section of the 9th nerve. In a case described by Erickson[4] in 1936, paroxysmal pain deep in the ear, without a demonstrable pharyngeal trigger point, was relieved by intracranial section of the 9th nerve. Glossopharyngeal neuralgia with pain restricted to the ear at the onset, and only later in its course associated with pharyngeal pain, has been reported by Martin[9] and by Jefferson.[6] In the case we wish to present, pain was restricted to the ear, and paresthesia without pain occurred in the region supplied by the mandibular branch of the fifth nerve.

For glossopharyngeal neuralgia to be accompanied by cardiac dysrhythmia and syncope or by other acute cerebral symptoms is unusual but not rare. Riley and his associates[15] first described patients whose glossopharyngeal neuralgia was combined with cardiac arrest and syncope, but did not report an operation. Ray and Stewart[12] told of a similar case and were the first to report cure of the condition by intracranial section of the 9th nerve. Karnosh *et al.*,[7] in 1947, described a case in which glossopharyngeal neuralgia was associated with cardiac arrest and dizziness. Roulhac and Levy[16] reported a case in which bradycardia, syncope and convulsions were associated with glossopharyngeal neuralgia. Richburg and Kern,[14] in a similar case, were able to dissociate the cardiac signs and secondary cerebral symptoms from glossopharyngeal pain by the use of atropine. Thomson,[19] in 1954, reported a case in

* The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

which neuralgia, apparently in the region supplied by the mandibular division of the 5th nerve, occurred in association with convulsive episodes, but without demonstrable changes in cardiac or vascular function. Pain was relieved by section of the 9th nerve, but not by partial section of the sensory root of the 5th.

REPORT OF CASE

The patient, a white man, 43 years of age, a tool and die maker, registered at the Mayo Clinic on Jan. 18, 1956. He complained of severe paroxysmal pain in the left ear. He had been well until April, 1950, when he had begun to have constant pain in his throat, more on the left side. This had been interpreted as a symptom of infection of the upper part of the respiratory tract. In spite of medication, this pain had persisted for a month. In May, 1950, while swallowing a pill, he had experienced paroxysmal pain in the ear. This was the first attack in a series. The pain had recurred from 1 to 30 times per day. The series of attacks lasted 3 weeks. Following it the man had been free of attacks for 13 months. From 1951 to 1955, he had had 9 episodes at intervals of approximately 3 months. At this stage there had been pain deep in the left ear canal, precipitated usually by swallowing either solids or liquids, occasionally by talking or brushing the teeth, or by pressure deep in the left ear canal. On many occasions, however, there had not appeared to be any precipitating cause.

The character and frequency of the attacks changed somewhat with the passage of time. Initially, the attacks had consisted of sudden, severe, momentary paroxysms of pain, each of increasing intensity, deep within the ear, recurring over a period of 20 to 30 seconds. These had been followed by an intense, painful, burning sensation lasting 2 to 3 minutes. On infrequent occasions the patient had experienced a tingling sensation anterior to the left ear and along the lower margin of the left mandible to the mandibular symphysis. The last episode of such attacks had begun in the fall of 1955 and continued at weekly intervals until Jan. 10, 1956. On this date, while eating, the patient had experienced 3 or 4 sudden stabbing pains in the left ear, and without warning had lost consciousness for 1 to 2 minutes. From then on he had experienced an increasing frequency of attacks of pain in the ear. On several occasions he had injured himself by falling against objects during a sudden loss of consciousness; however, he had never bitten his tongue, become incontinent, cried out, nor made convulsive movements.

On admission the patient was obviously fatigued and irritable. He had confined himself to bed because of the syncopal attacks and had not eaten for several days. During the day of admission, he had 12 to 15 attacks of pain, in many of which he lost consciousness. On several occasions partial or complete attacks were observed. After swallowing or breathing deeply, the patient would complain of 3 to 4 stabbing pains deep in the left ear and he would grimace terribly. His eyes would roll back; he would become pale, sweat and lose consciousness for 15 to 20 seconds. During these attacks his pulse was slowed, often becoming imperceptible.

General physical examination gave negative results. The blood pressure was 130 mm. of mercury systolic and 76 diastolic; the pulse was regular at a rate of 78 per minute. There was a suggestion of droop to the left eyelid and the left corner of the mouth; the patient attributed this to "favoring" that side in an attempt to avoid precipitating pain. Otherwise, the findings of neurologic examination were negative. Routine tests of blood and urine gave evidence of nothing extraordinary. Roentgenograms of the head, including views of the base of the skull, did not indicate the presence of abnormalities. Tests of vestibular function gave negative results. Pressure on the carotid sinus did not cause syncope or the pain typical of the attack. An electroencephalogram was negative.

To precipitate an attack while another electroencephalogram was being recorded, the patient was asked to induce hyperventilation; this produced the expected pain followed by syncope. During the painful part of the attack there was a generalized slowing of the frequency of waves on the electroencephalogram. When the patient became unresponsive, the record became markedly flattened. After 15 seconds the patient began to recover. Coinci-

dentally, on the electroencephalogram another short phase of generalized slowing was recorded and then the waves returned to the normal state.

On another occasion the patient was asked to drink a glass of water while both an electroencephalogram and an electrocardiogram were recorded. Immediately upon swallowing, he had extreme pain, during which the electrocardiogram gave evidence of an irregular and slow pulse (Fig. 1). On this occasion syncope did not occur and there were no electroencephalographic changes. On the third occasion, the tonsillar region on the left side was stimulated, and this produced severe pain in the left ear, typical of the patient's presenting complaint.

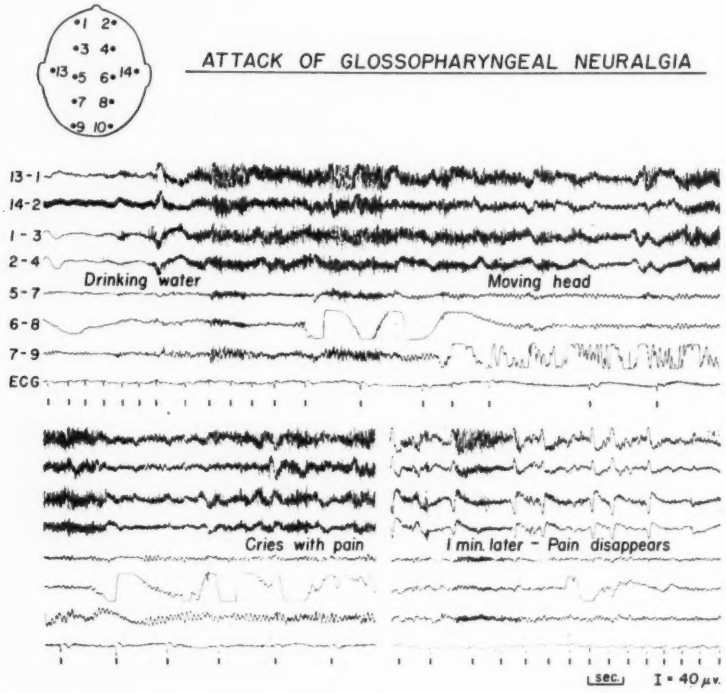


FIG. 1. Simultaneous electroencephalographic and (bottom line) electrocardiographic recordings. At the onset of the attack of pain there is marked cardiac irregularity with an arrest of ventricular contraction for almost 3 seconds. When the pain is present, the heart rate slows to about 40 beats per minute, returning to normal as the attack subsides.

Anesthetization of the left tonsillar fossa by cocaine prevented for 20 minutes any painful results of further stimulation.

On January 24, left occipital craniectomy was performed for partial glossopharyngeal neuralgia. The dura mater was incised and reflected, and the occipital lobe was retracted. The 9th cranial nerve was identified and was found to be rather thicker than usual. This nerve and a rostral fourth (estimated) of the vagal fibers were sectioned. After this sectioning, the patient's pulse rate rose from 80 to 100 per minute. On the patient's return to the ward, his pulse rate was recorded as 120, and it remained between 100 and 110 for the first 4 postoperative days, after which it returned to normal. There was no change in his blood pressure during or after the operation.

After the operation, the patient was completely relieved of his pain. He was able to swallow without difficulty and stimulation of the tonsillar fossa did not produce an attack. Unfortunately, the area of anesthesia could not be outlined reliably because of an active gag reflex. The patient remained free of pain 6 months after the operative procedure, the time of writing of this report.

COMMENT

Obviously, a patient having paroxysmal, incapacitating pains may not always be able to localize accurately their site of origin and region of spread. However, pain in the ear seems to occur in association with glossopharyngeal neuralgia in three sequences: (1) pain beginning in the pharynx and spreading to the ear, which is common;[21] (2) pain beginning in the ear and spreading to the pharynx and base of the tongue, which is uncommon,[11] and (3) pain restricted to the ear, which is rare.[21] Two cases in which pain was restricted to the ear in association with glossopharyngeal neuralgia have been reported, and neither patient had a pharyngeal trigger point. Reichert[13] designated this condition as "partial" or "tympanic plexus" neuralgia, and attributed the syndrome to neuralgia of the tympanic branch of the glossopharyngeal nerve. In our case, pain was restricted to the ear after the fuller development of the complaint, although—fortunately for the purpose of diagnosis—a pharyngeal trigger point was present. Several instances have been reported in which pain was confined to the ear for some time before pharyngeal pain became a feature. In a case reported by Jefferson,[6] pain had been restricted to the ear for 30 years before a pharyngeal component became obvious. Possibly, in time, our patient also would have had typical pharygneal pain.

The glossopharyngeal branches of the tympanic plexus are connected to the mandibular division of the 5th nerve by two pathways. 1. A tympanic branch joins intracranially the small superficial petrosal nerve, which in turn makes connection with the otic ganglion. Final connections are made from the otic ganglion to the auriculotemporal branch of the mandibular division of the 5th nerve. 2. An extracranial branch of the glossopharyngeal joins the digastric division of the facial nerve. The fibers then pass along the chorda tympani to join the lingual nerve.

Pain in the region supplied by the 5th cranial nerve has been reported to occur in cases in which the diagnosis of glossopharyngeal neuralgia seems certain. Peet[11] reported 5 cases of glossopharyngeal neuralgia in which pain also occurred in the region of distribution of the trigeminal nerve, although not simultaneously. In 2 of these cases the pain was in the region supplied by the 1st or 2nd division of the 5th nerve where there could be no doubt as to the trigeminal distribution of the pain. Peet[11] suggested the possibility of two separate "diseases." However, Karnosh *et al.*[7] expressed the belief that pain in the region supplied by the 5th nerve might be caused by discharges emanating from the glossopharyngeal nerve along parasympathetic branches, and furthermore, that the pain might be relieved by section of the glossopharyngeal nerve. Our patient occasionally had paresthesia in the mandibular division of the 5th nerve. Recognition of the delusive character of this phenomenon is important, as surgical measures directed to the mandibular division of the 5th nerve will be ineffective in relieving the symptoms.

The history given by our patient made it apparent that another trigger point in the ear may have functioned occasionally. When such a trigger zone is suspected, but cannot be demonstrated at the time of examination, or when pain in the ear is predominant, neuralgia of five separate nerves must be considered.[3] These are: the tympanic branches of the glossopharyngeal, the auriculotemporal branch of the

trigeminal, the small sensory component of the facial, the auricular branch of the vagus, and the upper fibers of the cervical nerves. When other evidence of glossopharyngeal neuralgia is present, the possibility that the auricular branch of the vagus is also active as a trigger point is of practical importance, as pain may recur or persist if the upper fibers of the vagus are not sectioned at the time of section of the glossopharyngeal.[10,18] Dandy[3] routinely sectioned these fibers, but for another reason; namely, that sensitivity sometimes remained in the pharyngeal region after section of the glossopharyngeal nerve, a fact which suggested that in such cases the upper fibers of the vagus may have supplied this region. Furthermore, as an anatomic variation, the glossopharyngeal nerve may enter the brain stem as two roots.

Symptoms caused by cerebral anoxemia consequent to disturbance of cardiac or vascular function in association with glossopharyngeal neuralgia may be expected to occur more often than has been reported; publication of reports of 9 cases since 1942 indicated this phenomenon cannot be considered rare. Fibers from the carotid sinus, constituting the afferent limb of the carotid sinus reflex, join the glossopharyngeal nerve before its entrance into the brain stem; subsequently, they make central connections with the vagus. The latter nerve supplies the efferent limb of this reflex arc. Abnormal states in the glossopharyngeal nerve might, therefore, be expected to initiate cardiovascular disturbances, with secondary cerebral hypoxia resulting in syncope, such as occurred in our case. An increase in pulse rate following operation is attributed by Ray and Stewart[12] to release of vagal centers after interruption of the afferent pathways in the carotid sinus reflex. A rise in blood pressure is to be expected when the glossopharyngeal nerve is sectioned bilaterally.

SUMMARY

In a case encountered at the Mayo Clinic, pain was restricted to the ear and a pharyngeal trigger point was identifiable. Attacks of pain often were accompanied by syncope. The condition was identified as partial glossopharyngeal neuralgia. Complete relief followed intracranial section of the 9th cranial nerve and a rostral fourth (estimated) of the vagal fibers. In our case, it seems likely that the abnormal condition of the glossopharyngeal nerve initiated cardiovascular disturbances and secondary cerebral hypoxia and that the reflex pathway consisted of fibers from the carotid sinus that join the glossopharyngeal nerve and subsequently make central connections with the vagus.

REFERENCES

1. Adson, A. W. The surgical treatment of glossopharyngeal neuralgia. *Arch. Neurol. Psychiat., Chicago*, 1924, *12:* 487–506.
2. Dandy, W. E. Glossopharyngeal neuralgia (tic douloureux). Its diagnosis and treatment. *Arch. Surg., Chicago*, 1927, *15:* 198–214.
3. Dandy, W. E. The brain. In: *Lewis' practice of surgery.* Hagerstown, Md.; W. F. Prior Co., Inc., 1945, *12:* 187–191.
4. Erickson, T. C. Paroxysmal neuralgia of the tympanic branch of the glossopharyngeal nerve. Report of a case in which relief was obtained by intracranial section of the glossopharyngeal nerve. *Arch. Neurol. Psychiat., Chicago*, 1936, *35:* 1070–1074.
5. Harris, W. Persistent pain in lesions of the peripheral and central nervous system. *Brain*, 1921, *44:* 557–571.
6. Jefferson, G. Glosso-pharyngeal neuralgia. *Lancet*, 1931, *2:* 397–399.
7. Karnosh, L. J., Gardner, W. J., and Stowell, A. Glossopharyngeal neuralgia: physiologic considerations of the role of the ninth and tenth cranial nerves. Report of cases. *Trans. Amer. neurol. Ass.*, 1947, 205–207.

8. Love, J. G. Diagnosis and surgical treatment of glossopharyngeal neuralgia. *Surg. Clin. N. Amer.*, August 1944, 959–962.
9. Martin, J. P. A case of glossopharyngeal neuralgia. *Brit. med. J.*, 1931, *1:* 533–534.
10. Pastore, P. N., and Meredith, J. M. Glossopharyngeal neuralgia. Tic douloureux of the ninth cranial nerve. *Arch. Otolaryng., Chicago*, 1949, *50:* 789–794.
11. Peet, M. M. Glossopharyngeal neuralgia. *Ann. Surg.*, 1935, *101:* 256–268.
12. Ray, B. S., and Stewart, H. J. Glossopharyngeal neuralgia: a cause of cardiac arrest. *Amer. Heart J.*, 1948, *35:* 458–462.
13. Reichert, F. L. Neuralgias of the glossopharyngeal nerve with particular reference to the sensory, gustatory and secretory functions of the nerve. *Arch. Neurol. Psychiat., Chicago*, 1934, *32:* 1030–1037.
14. Richburg, P. L., and Kern, C. E. Glossopharyngeal neuralgia with syncope and convulsions. *J. Amer. med. Ass.*, 1953, *152:* 703–704.
15. Riley, H. A., German, W. J., Wortis, H., Herbert, C., Zahn, D., and Eichna, L. Glossopharyngeal neuralgia initiating or associated with cardiac arrest. *Trans. Amer. neurol. Ass.*, 1942, 28–29.
16. Roulhac, G. E., and Levy, I. Glossopharyngeal neuralgia associated with cardiac arrest and convulsions. *Arch. Neurol. Psychiat., Chicago*, 1950, *63:* 133–139.
17. Sicard, R., and Robineau. Algie vélo-pharyngée essentielle. Traitement chirurgical. *Rev. neurol.*, 1920, *36:* 256–257.
18. Spurling, R. G., and Grantham, E. G. Glossopharyngeal neuralgia. *South. med. J.*, 1942, *35:* 509–512.
19. Thomson, J. L. Glossopharyngeal neuralgia accompanied by unconsciousness. *J. Neurosurg.*, 1954, *11:* 511–514.
20. Weisenburg, T. H. Cerebello-pontile tumor diagnosed for six years as tic douloureux. The symptoms of irritation of the ninth and twelfth cranial nerves. *J. Amer. med. Ass.*, 1910, *54:* 1600–1604.
21. Wilson, S. A. K. Neurology. *Baltimore, Md.: Williams & Wilkins Co.*, 1955, 2nd ed., *1:* 435.

BRAIN ABSCESS IN TWO PATIENTS WITH CONGENITAL CARDIAC DISEASE

E. Ley, M.D.,* S. Obrador, M.D.† and E. Ley, Jr., M.D.‡

Neurosurgical Service, Central Hospital of the Red Cross, and Neurosurgical Section, Institute of Medical and Clinical Research, Madrid, Spain

(Received for publication November 26, 1956)

The so-called paradoxic brain abscess in congenital heart disease apparently is encountered rarely in neurosurgical practice, for very few examples have been recorded in the literature. Also the surgical results obtained have been poor. In fact, in a review of the neurosurgical literature of the last 10 years, only 6 cases were found in which this condition was reported as cured.

Smolik *et al.*,[5] in 1946, were the first to report a brain abscess of this type cured by surgical drainage and sulfonamides.[2] Two other cases of successful removal of the abscess under the protection of antibiotics were reported in 1951 by Cohen *et al.*[2] and Beller.[1] Ingraham and Matson[3] mentioned 2 instances in which brain abscess associated with tetralogy of Fallot was successfully removed. Finally, in 1956, Lafon *et al.*[4] recorded another patient with the same condition who recovered after excision of the abscess.

Within the last few months, we have operated upon 2 patients with brain abscess associated with congenital heart disease of the type of the tetralogy of Fallot. These 2 cases are the only examples of this condition observed in our neurosurgical practice during the last 15 years.

The rarity of this brain lesion, together with the isolated and rather exceptional reports of surgical cure, justify, in our view, the publication of a summary of the clinical records of our 2 patients.

CASE REPORTS

Case 1. M.C.B., a girl 8 years of age, was admitted to one of our Neurosurgical Services (E.L.) of the Central Hospital of the Red Cross on May 29, 1956. When the patient was 1 month old it was found that she had a cardiac bruit. Cyanosis and breathlessness were noted when she began to walk at the age of 1 year. A diagnosis of tetralogy of Fallot was established. The cardiac symptoms gradually increased and Blalock's operation was performed when she was 6 years old. She was greatly improved subsequently and had only minor illnesses, i.e. whooping cough. Three months before admission she suffered a cold with cough and some nasal secretion. About the same time she had a tooth extracted without any obvious complication. Ten days later her temperature was elevated and a slight epileptiform attack occurred, with twitchings in the right side of the face for several minutes but without loss of consciousness. The fever continued and the attacks recurred, frequently spreading to the right arm. In spite of treatment with antibiotics there developed weakness of the right arm, right side of the face, and right leg. At this time she had headaches accompanied by vomiting and later by somnolence.

Examination. The patient was in poor condition, cyanotic and somewhat lethargic. Positive findings were bilateral papilledema, slight low right facial weakness, right hemiparesis,

* Neurosurgical Service, Central Hospital of the Red Cross, Madrid, Spain.

† Martinez Campos, 11, Madrid, Spain.

‡ Fernando el Catolico, 14, Madrid, Spain.

more marked in the arm, with hypertonia, increased tendon jerks and positive Babinski and Hoffmann signs. There was a cardiac murmur on auscultation. The only positive laboratory finding was leucocytosis (14,200) with some increase of neutrophils (72 per cent).

Left carotid arteriography confirmed the presence of an expansive lesion in the left parietal region.

1st Operation. Through a left parietal burr hole an abscess was tapped and 45 cc. of pus were removed. Penicillin and streptomycin were injected into the abscess cavity.

Bacteriological study of the pus and its culture failed to demonstrate any micro-organisms.

Course. Though the child improved immediately after operation and received antibiotics, on the 9th postoperative day the hemiparesis increased and she became stuporous. Lumbar puncture showed a spinal fluid pressure of 280 mm. of water. The abscess was tapped again and 35 cc. of pus were evacuated and more antibiotics were injected locally. Bacteriological studies of the pus again revealed no organisms. In spite of some improvement in the patient's condition, the cerebrospinal fluid pressure continued to increase (over 300 mm. of water) and another tap of the abscess was performed, 25 cc. of sterile pus being obtained.

2nd Operation. Under general anesthesia a lateral osteoplastic flap was carried out, nearly 3 weeks after the first brain puncture. A subcortical parietal abscess with a definite capsule was totally removed. Penicillin and streptomycin were instilled in the operative cavity of the brain and the wound was closed without drainage.

Postoperative course was uneventful except for a reaction to a blood transfusion 24 hours after surgery. The child was discharged 10 days after operation. She had no symptoms of increased intracranial pressure but the right hemiparesis persisted. This has improved steadily during the 4 months since operation.

Case 2. M.C.M.J., a girl 7 years of age, was admitted to one of the Medical Services (Dr. Barreda) of the Institute of Medical and Clinical Research (Director Prof. Jimenez Diaz) on July 17, 1956. Shortly after birth her mother noticed a cyanotic coloration of the distal portions of the extremities and of the tongue. She had occasional difficulty in breathing. These symptoms gradually increased, becoming more apparent when the girl tried to play and run with other children. At the same time the tips of the fingers slowly became deformed and had the typical shape of drumsticks.

Twenty days before her admission to the clinic for consideration of surgical treatment of her cardiac disease, she began to have headaches and vomiting. Her temperature was elevated and a diagnosis of possible meningitis was made by several physicians. She was treated with antibiotics.

Examination. The patient was fairly well developed, but rather pale, and cyanosis, especially marked on the lips, was evident. A strong systolic murmur was heard in auscultation of the heart. The edge of the liver was felt just below the costal margin. Typical clubbing of the fingers of both hands was present.

Roentgenograms revealed hypertrophy of the right ventricle of the heart, a concave left border of the heart secondary to disappearance of the pulmonary conus, and extreme dextroposition of the aorta in the right anterior oblique view. There was a right axis deviation in the electrocardiogram. The clinical diagnosis made by the Department of Cardiovascular Surgery (Dr. Castro Fariñas) was Fallot's tetralogy.

Routine laboratory studies showed anemia, normal white blood cells and increased sedimentation rate (index 48.5). Culture of the blood (Schottmüller's method) was negative.

On the first examination made by the Neurosurgical Section of the Institute, directed by one of us (S.O.), the child was found to be very stuporous. Positive neurological findings were slight rigidity of the neck, bilateral papilledema, paresis of the right 6th cranial nerve and discrete right hemiparesis with a tendency to a Babinski sign on that side. Although study of sensibility was difficult because of the patient's condition, some alteration of postural sense was obvious in the right hand and foot.

Operations and Course. An exploratory puncture in the left parieto-occipital region of the brain through a burr hole disclosed a deep abscess and 80 cc. of pus were aspirated. Penicillin

and streptomycin were injected locally. During the following 10 days two more taps of the abscess were carried out, 30 and 7 cc. of pus being removed. The child improved with this treatment and systemic administration of antibiotics, but about 2 weeks later fever and headaches recurred. Another puncture of the abscess failed to obtain much pus and the resistance of the capsule was then very definite. The injection of a few cc. of Pantopaque into the cavity of the abscess and subsequent roentgenograms revealed the extension and depth of the lesion.

Bacteriological Studies (Dr. Ales). The culture of the pus showed a late growth (2–3 days) of a gram negative coccus-bacillus which can be included in the *Parvobacteriaceae*, group *Pasteurelleae*, type *Actinobacillus lignieresi*. Sensibility studies of this micro-organism demonstrated that Terramycin, aureomycin, Chloromycetin and Tetracyclin were the more active antibiotics.

Course. The initial improvement of the child after the first punctures of the abscess was not sustained. She continued to complain of headaches, her temperature was elevated, and there were periods of obnubilation and stupor. The papilledema and some degree of right hemiparesis persisted and she also showed slight meningeal signs. The cerebrospinal fluid was, however, normal.

4th Operation. A left parieto-occipital bone flap was carried out under general anesthesia about 7 weeks after the first puncture of the abscess. Through a cortical incision a very large and deeply situated abscess was exposed and its capsule, very irregular in shape, was dissected from the white matter until it was completely removed. In some places the capsule was very hard and resistant, but elsewhere it appeared to be very thin and was broken during the dissection. At this point a fair amount of pus escaped. The lateral ventricle was opened because of the deep location of the abscess, the choroid plexus being coagulated partially. The residual cavity left by the excision of the abscess measured about 7×5 cm. in the anteroposterior and coronal planes. Antibiotics were instilled locally and the wound was closed without drainage.

Postoperative course was quite good. Several lumbar punctures were necessary because the intracranial pressure was moderately high. There was an increase of cells in the cerebrospinal fluid during the first weeks after operation, but this and other less important changes slowly cleared up. Terramycin was given during the first 2 postoperative weeks. A very slight decrease of motor power and some alteration of postural sense in the right limbs were the only positive neurological findings at the time of her discharge, 3 weeks after operation. The symptoms of increased intracranial pressure had completely subsided and the patient's general condition was good at that time. Unfortunately this child presented later a sudden and unexpected recurrence of the abscess and died 5 months after operation in spite of immediate cerebral puncture with evacuation of a large amount of pus.

SUMMARY

Two children, 7 and 8 years old, suffering from Fallot's tetralogy, one of them having been operated upon 2 years previously (Blalock's operation) and the other still being studied, exhibited symptoms of increased intracranial pressure and focal signs indicating a lesion in the left cerebral hemisphere. One had no history of an obvious infection, and the other had had only a slight cold and extraction of a tooth 10 days before onset of symptoms. The clinical diagnosis of cerebral abscess associated with congenital heart disease was confirmed by puncture of the brain. A very rare coccus-bacillus was isolated from the pus in 1 of the cases. In the other the pus was sterile. After several punctures the abscesses were totally removed under the protection of locally and systemically administered antibiotics. An excellent immediate result was obtained in both cases although one of the patients died with sudden and unexpected recurrence of the abscess several months afterwards.

The previously reported examples of surgical cure of brain abscesses associated

with congenital cardiac disease are so exceptional that only 6 other cases were found in the neurosurgical literature.

REFERENCES

1. Beller, A. J. The syndrome of brain abscess with congenital cardiac disease. Report on a case with complete recovery. *J. Neurosurg.*, 1951, *8:* 239–243.
2. Cohen, I., Bergman, P. S., and Malis, L. Paradoxic brain abscess in congenital heart disease. *J. Neurosurg.*, 1951, *8:* 225–231.
3. Ingraham, F. D., and Matson, D. D. Neurosurgery of infancy and childhood. *Springfield, Ill.: Charles C Thomas*, 1954, xvii, 456 pp.
4. Lafon, R., Labauge, R., Cadilhac, J., Vlaovitch, B., and Suire, A. Abcès cérébraux à corynebacterium liquefaciens, complication d'une pentalogie de Fallot, guéris par interventions neurochirurgicales successives. *Rev. neurol.*, 1956, *94:* 386–394.
5. Smolik, E. A., Blattner, R. J., and Heys, F. M. Brain abscess associated with congenital heart disease. Report of a case with complete recovery. *J. Amer. med. Ass.*, 1946, *130:* 145–147.

MULTIPLE PRIMARY NEOPLASMS OF THE CENTRAL NERVOUS SYSTEM

REPORT OF A CASE OF SPINAL EPENDYMOMA AND AN INTRACRANIAL MENINGIOMA. INTERVAL OF FOURTEEN YEARS*

JAMES GREENWOOD, JR., M.D., AND T. H. MCGUIRE, M.D.
Methodist Hospital and Baylor University College of Medicine, Houston, Texas

(Received for publication December 13, 1956)

Multiple neoplasms of the central nervous system are no longer pathological curiosities, but in most reported cases the tumors arose from the same germ layer. Courville[2] in 1936 collected from the literature 113 cases of multiple gliomas of the brain, to which he added 21 of his own. The case of multiple intracranial meningioma, reported by Echols[5] in 1941, brought to 53 the total up to that time. Two cases of combined acoustic neurinoma and meningioma were reported by Davidoff and Martin.[4] Cushing and Eisenhardt[3] had formerly called attention to the frequent association of tumors of the meninges with acoustic neoplasms, and it was the feeling of Kernohan[9] that multiple meningioma and neurofibroma are genetically related.

List[10] reported a case in which he removed four meningiomas from the region of the foramen magnum and the upper cervical cord. The patient of Rand[12] had separate spinal meningiomas at T3 and T6.

Alexander[1] was able to find only 4 cases in which an intracranial meningioma was reported in association with a glioma of the cerebral hemisphere. To these he added 1 case of his own. Since that time Gass and Van Wagenen[7] have reported a meningioma adjacent to an oligodendroglioma, and a patient of Feiring and Davidoff[6] had a meningioma in association with a glioblastoma multiforme. The 1940 edition of Wilson's[13] textbook of neurology collected from the literature 11 cases of multiple heterogeneous intracranial new growths.

Kaelber[8] pointed out the possibility of dissimilar lesions of the cerebrospinal axis by reporting his case of an intracranial meningioma associated with syringomyelia. Poser[11] recently reviewed the literature and showed the frequent association of syringomyelia with intramedullary neoplasms of the spinal cord. Syringomyelia is in this case a complication.

Because of the relatively few reported cases of heterogeneous neoplasms of the central nervous system, we are reporting our experience with a case of intracranial meningioma and a spinal ependymoma.

CASE REPORT

This was our first case of ependymoma with complete recovery.

Mrs. C.N., a 44-year-old, right-handed housewife, was first seen in August of 1941 with a 10-month history of progressive weakness in both legs and difficulty in walking. There was some mild discomfort in the thoracolumbar region on weight-bearing and numbness in both legs, which had begun 3 or 4 months after the onset of motor weakness and had ascended to the upper abdomen. For the past 4 or 5 months before examination, she had been unable to appreciate changes in temperature in the lower extremities. There was no significant abnormality of bladder or bowel.

* Read before Southern Neurosurgical Society, Jacksonville, Florida, March 24, 1956.

Examination, Aug. 13, 1941. There was marked weakness in both lower extremities and she was unable to walk without assistance. There were increased reflexes in both legs, transient ankle clonus, and bilateral Babinski sign, with no involvement of the upper extremities. While touch and position sense were fairly good in the lower extremities, loss of sensation of pain, temperature, vibration and stereognosis extended upward to the level of the 5th rib.

Myelography revealed a complete block at the upper border of the 7th thoracic vertebra.

Operation. Laminectomy, extending from C7 through T6 inclusive, showed moderate swelling of the cord, greatest at the level of T5. The spinal cord was incised in the midline and a dark red, sharply circumscribed tumor, attached only in the region of the central canal, was carefully removed. The tumor extended throughout the length of the exposure and the estimated weight was 20 gm.

Microscopic Examination (Fig. 1). The tumor was composed of spindle cells that varied in shape and size, with hyperchromatic nuclei which were highly granular. Mitotic figures were present but were not abundant. There was a tendency to rosette arrangement. Diagnosis: Ependymoma.

Course. Her condition following surgery was definitely worse, but she was able to walk within 6 months, and at the end of the year walked perfectly, although she tired easily. Pain and other forms of sensation recovered completely, except for some hypesthesia in the sacral segments on the left. Reflexes were essentially normal.

Interval History. In 1948, the patient had a thyroidectomy with removal of a benign adenoma, and in 1950 a hysterectomy for a uterine fibroid. Gait had continued to be normal and she had no symptoms until 1953.

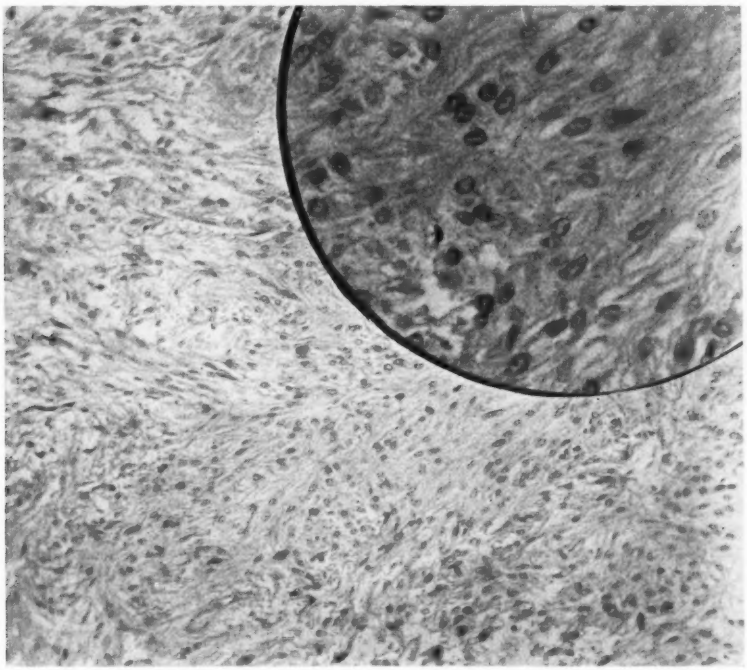


FIG. 1. Photomicrographs of ependymoma, intramedullary, C7-T6. Total removal in 1941, with complete relief of symptoms.

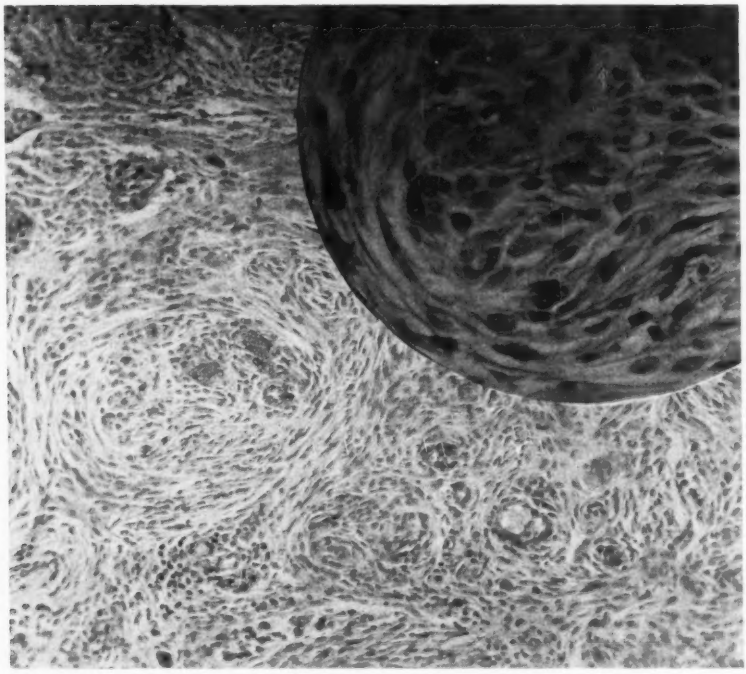


FIG. 2. Photomicrographs of meningioma of right sphenoid wing, removed in 1955, 14 years after spinal operation.

Readmission. She was re-examined in the hospital in July 1955, at which time there was a history of throbbing headaches, often localized to the right orbital area, which had begun 19 months before. Examination revealed the patient to be in semicoma with left-sided weakness, cogwheel rigidity, positive Hoffmann sign, Babinski sign, and increased deep reflexes. There was a mild papilledema with moderate nuchal rigidity, and a spinal puncture had shown a pressure of 370 mm. of cerebrospinal fluid. Electroencephalography revealed a slow wave focus in the right frontotemporal area.

Operation, July 12, 1955. Ventriculography demonstrated a shift to the left with typical temporal lobe findings. At surgery, immediately following, there was an area of softening in the tip of the right temporal lobe, and exploration with a cannula disclosed a large sphenoidal ridge meningioma. This was completely removed, and its attachment to the medial portion of the sphenoidal ridge was less vascular than in similar tumors.

Course. In spite of this, postoperative clots were evacuated on the 1st and 2nd days following surgery, after which her recovery was uneventful except for partial left homonymous field defect. She was able to resume her household duties within a short time.

Microscopic Examination (Fig. 2). The tumor was cellular, and the cells were uniform in size and shape, most of them having a spindle configuration with a tendency to whorl formation. A moderate amount of collagen was seen, and the vascular supply was abundant. Weight of the tumor was 40 gm. Diagnosis: Meningioma.

SUMMARY AND CONCLUSIONS

1. The literature on the occurrence of multiple neoplasms of the central nervous system has been reviewed.

2. A case has been reported, recording the occurrence of a spinal cord ependymoma and a sphenoidal ridge meningioma in the same patient, with an interval of 14 years between surgical removal of the tumors.

3. Multiple neoplasms of the central nervous system are not uncommon, but usually arise from the same germ layer. Instances of occurrence in heterogeneous germ layers have been quite rare, as indicated by the few case reports. This case, we believe, is the first instance of intracranial meningioma and spinal ependymoma occurring in the same patient.

REFERENCES

1. Alexander, W. S. Multiple primary intracranial tumours. Meningioma associated with a glioma; report of a case. *J. Neuropath. exp. Neurol.*, 1948, *7:* 81–88.
2. Courville, C. B. Multiple primary tumors of the brain. Review of the literature and report of twenty-one cases. *Amer. J. Cancer*, 1936, *26:* 703–731.
3. Cushing, H., and Eisenhardt, L. Meningiomas. Their classification, regional behaviour, life history, and surgical end results. *Springfield, Ill.: Charles C Thomas*, 1938, xiv, 785 pp.
4. Davidoff, L. M., and Martin, J. Hereditary combined neurinomas and meningiomas. *J. Neurosurg.*, 1955, *12:* 375–384.
5. Echols, D. H. Multiple meningioma. Removal of ten intracranial tumors from a patient. *Arch. Neurol. Psychiat., Chicago*, 1941, *46:* 440–443.
6. Feiring, E. H., and Davidoff, L. M. Two tumors, meningioma and glioblastoma multiforme, in one patient. *J. Neurosurg.*, 1947, *4:* 282–289.
7. Gass, H., and van Wagenen, W. P. Meningioma and oligodendroglioma adjacent in the brain. Case report. *J. Neurosurg.*, 1950, *7:* 440–443.
8. Kaelber, W. W. Dissimilar lesions of the nervous system: brain tumor and syringomyelia. *J. Neuropath. exp. Neurol.*, 1952, *11:* 79–82.
9. Kernohan, J. W. Tumors of the spinal cord. *Arch. Path.*, 1941, *32:* 843–883.
10. List, C. F. Multiple meningiomas. Removal of four tumors from region of the foramen magnum and upper cervical region of the cord. *Arch. Neurol. Psychiat., Chicago*, 1943, *50:* 335–341.
11. Poser, C. M. The relationship between syringomyelia and neoplasm. *Springfield, Ill.: Charles C Thomas*, 1956, ix, 98 pp.
12. Rand, R. W. Multiple spinal cord meningiomas. *J. Neurosurg.*, 1952, *9:* 310–314.
13. Wilson, S. A. K. Neurology. *Baltimore: Williams & Wilkins Co.*, 1940, *2:* 1219.

BRACHIAL VERTEBRAL ANGIOGRAPHY IN ADULTS

WILLIAM F. COLLINS, JR., M.D., HARRY W. SLADE, M.D.,* AND WILLIAM G. LOCKHART, M.D.
Division of Neurosurgery, School of Medicine, Western Reserve University, and University Hospitals, City Hospital, and Crile Veterans Administration Hospital, Cleveland, Ohio

(Received for publication October 18, 1956)

The difficulties involved in total cranial angiography have been emphasized by numerous authors.[2,4,5,7,9] Gould *et al.*[1] described a technique for vertebral angiography applicable to children. This consisted of retrograde injection of contrast material via the right brachial artery. They were unable to recommend the technique for adults because they felt the bolus of dye was diluted so that adequate contrast was difficult to obtain.

The purpose of this report is to present a modification of this technique which has enabled us to obtain consistently good films in adults. Since 1955, it has been used in 17 cases. The first of the series demonstrated filling of the posterior circulation but the contrast was not satisfactory. Following this, the changes in technique described below were made and in the remaining 16 cases the contrast was sufficient for diagnostic purposes.

TECHNIQUE

The technique used is basically that described by Gould *et al.*[1] Under local anesthesia the right brachial artery is exposed through a 3- to 4-cm. incision in the bicipital groove at the junction of the middle and distal thirds of the arm. A 3-cm. segment of artery is mobilized from the neurovascular bundle and umbilical tape is placed proximally and distally. With traction on the tape for hemostasis, a small longitudinal incision is made in the artery and a #12-gauge Robb angiograph cannula is placed in the artery. The cannula is secured by tying the proximal tape securely about the frame of the needle. A syringe with a #12-gauge opening is attached to the cannula and 25 to 40 cc. of contrast material are injected as rapidly as possible (in 2 to 3 seconds).

In addition to providing an easy means for visualization of the posterior circulation, it is possible, with compression of the left carotid, to secure a total cranial angiogram in which only the left carotid siphon below the circle of Willis remains unfilled. Three contrast media (30 per cent Hypaque, 35 per cent Diodrast, 30 per cent Renografin) have been used and, aside from the subjective burning reported by the patients injected with Diodrast, there has been no essential difference in the results obtained with these substances.

Films of the skull are made in lateral and Towne's views with submental vertical projection when indicated. The timing of the injections with multiple exposure technique is approximately the same as with a percutaneous carotid injection. When a single film is exposed, the exposure is delayed a fraction of a second beyond the usual carotid timing. At the close of the procedure, a single arterial silk suture is adequate for closing the arterial wound. The circulation of the hand has shown no signs of impairment and the radial pulse is usually palpable 48 hours after injection.

RESULTS

With the exception of the first case in the series, in which diagnostic contrast was not obtained, the results have been satisfactory when using the modifications outlined. In the series, 5 patients had spontaneous subarachnoid hemorrhage: 4 of these had negative bilateral carotid arteriograms and the vertebral arteriograms

* Present address: 2224 Washington Avenue, Waco, Texas.

were also negative; the remaining patient, whose case is outlined in detail below (Case 1, Fig. 1), was found to have an aneurysm of the basilar artery at the junction of the superior cerebral artery on the right. There were 5 cases of possible arteriovenous malformations, 4 of which were demonstrated. One of these is presented in detail below (Case 2, Fig. 2). In 5 cases of possible posterior fossa tumors arteriography did not prove helpful. One patient with a tumor of the 3rd ventricle was examined and the lack of displacement in the posterior circulation aided in the diagnosis of craniopharyngioma.

Case 1. H.St.C., a 48-year-old colored female, was admitted to University Hospitals on Jan. 8, 1956 with a history of sudden loss of consciousness 3 hours previously.

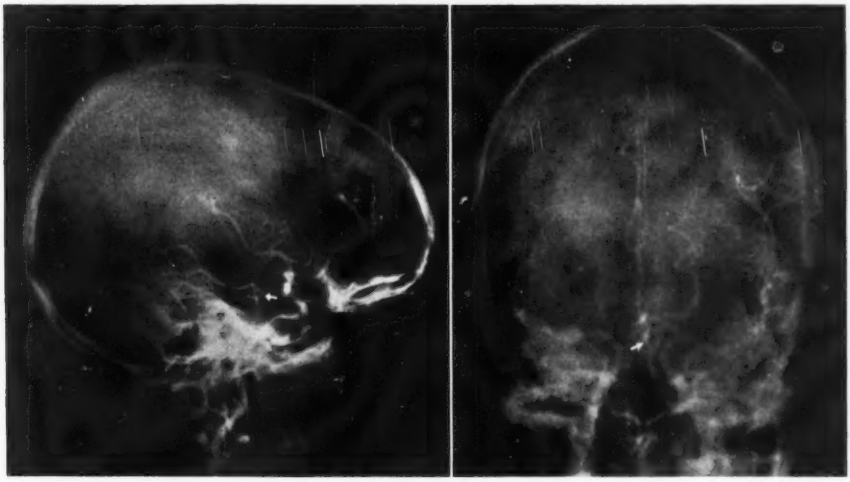


FIG. 1. *Case 1.* Aneurysm of the basilar artery.

Examination on admission revealed blood pressure of 208/95, pulse rate of 60, and respiratory rate of 24. Temperature was 37.8°C. The patient responded to noxious stimuli by decerebrate posturing; her pupils were pinpoint and fixed, and her neck was stiff. Lumbar puncture revealed grossly bloody fluid.

During the next 12 hours the patient's level of consciousness improved and only fragments of decerebration remained. Ten days after admission she was alert and cooperative. There was loss of upward gaze and she had a right hemiparesis without aphasia. On Jan. 20, 1956 a retrograde angiogram was performed, demonstrating an aneurysm of the basilar artery (Fig. 1). The patient was discharged improved on Jan. 28, 1956.

Case 2. B.W., a 29-year-old colored female, was admitted to City Hospital on Nov. 28, 1955 with a history of increasing headache, vertigo and ataxia for 1½ years. Three weeks prior to admission the patient had onset of blurred vision and diplopia.

Examination on admission revealed papilledema with right cerebellar signs. On Dec. 5, 1955 ventriculograms were read as consistent with a posterior fossa tumor and a suboccipital craniectomy revealed a vascular abnormality involving the right cerebellar hemisphere.

On Dec. 28, 1955 a retrograde angiogram was done demonstrating a large arteriovenous malformation of the right cerebellar hemisphere (Fig. 2); this malformation was considered not amenable to surgery. Exploration would have been obviated had this study been elected pre-operatively.

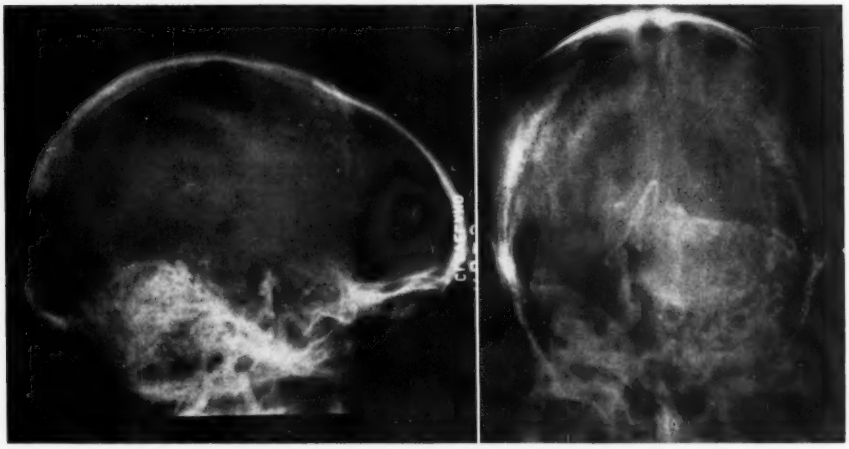


FIG. 2. *Case 2.* Large arteriovenous malformation of the right cerebellar hemisphere.

DISCUSSION

Previous methods of vertebral angiography,[2–4,6–8,10] while giving good diagnostic films, have either been technically difficult to perform, fraught with danger to neighboring structures, or complicated by the need for considerable manipulation in roentgen ray. The authors feel that with the modifications of using a #12-gauge arterial cannula and a syringe with a #12-gauge opening, the technique described by Gould *et al.*[1] has consistently given, in adults, diagnostic vertebral angiograms with no morbidity or mortality in the series presented.

SUMMARY

A series of vertebral angiograms in 17 cases, made by retrograde injection of the right brachial artery, is presented. The method used to insure good roentgen-ray visualization is described.

REFERENCES

1. GOULD, P. L., PEYTON, W. T., and FRENCH, L. A. Vertebral angiography by retrograde injection of the brachial artery. *J. Neurosurg.*, 1955, *12:* 369–374.
2. LINDGREN, E. Percutaneous angiography of the vertebral artery. *Acta radiol., Stockh.*, 1950, *33:* 389–404.
3. MONIZ, E., and ALVES, A. L'importance diagnostique de l'artériographie de la fosse postérieure. *Rev. neurol.*, 1933, *2:* 91–96.
4. NAMIN, P. Percutaneous vertebral angiography. *J. Neurosurg.*, 1954, *11:* 442–457.
5. PERESE, D. M., KITE, W. C., BEDELL, A. J., and CAMPBELL, E. Complications following cerebral angiography. *Arch. Neurol. Psychiat., Chicago*, 1954, *71:* 105–113.
6. RADNER, S. Vertebral angiography by catheterization. A new method employed in 221 cases. *Acta radiol., Stockh.*, 1951, suppl. 87, 134 pp.
7. ROWBOTHAM, G. F., HAY, R. K., KIRBY, A. R., TOMLINSON, B. E., and BOUSFIELD, M. E. Technique and the dangers of cerebral angiography. *J. Neurosurg.*, 1953, *10:* 602–607.
8. SCHAERER, J. P. Open indirect method of vertebral angiography. *J. Neurosurg.*, 1955, *12:* 487–494.
9. SUGAR, O., and BUCY, P. C. Some complications of vertebral angiography. *J. Neurosurg.*, 1954, *11:* 607–615.
10. SUGAR, O., HOLDEN, L. B., and POWELL, C. B. Vertebral angiography. *Amer. J. Roentgenol.*, 1949, *61:* 166–182.

NEW INSTRUMENT

FRANK H. MAYFIELD, M.D.*
Cincinnati, Ohio

(Received for publication November 1, 1956)

Removal of calcified bony spurs from the cervical area (spondylosis) is facilitated by the use of a down-biting curette which utilizes the principles of an adenotome and a lathe, as illustrated in Fig. 1.

The instrument combines a large firm handle, which can be grasped with both hands, and a small cutting surface. The instrument is provided in sets of three, with the relationship of the cutting surface to the handle varied. With adequate exposure,

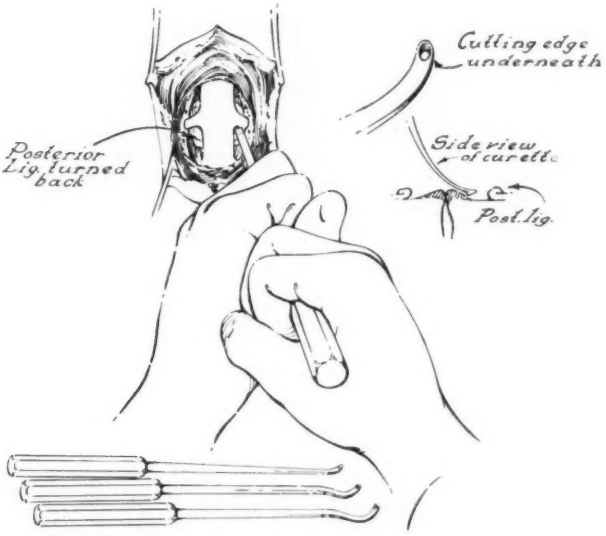


FIG. 1

bony ridges in the root canal, and indeed those that traverse the spinal canal, can be shaved off in small bits. The instrument is passed between the dura mater and the bony ridge without other retraction, and then is grasped firmly in both hands while resting the wrists on the margins of the wound and cutting with short strokes by closing the fists.

The instrument is being produced by the Kees Surgical Specialty Company.

* 506 Oak Street, Cincinnati 19, Ohio.

A MODIFICATION OF TORKILDSEN'S VENTRICULOCISTERNOSTOMY

CARL J. GRAF, M.D., AND WALLACE B. HAMBY, M.D.
University of Buffalo School of Medicine and the Buffalo General Hospital, Buffalo, New York

(Received for publication November 7, 1956)

Arne Torkildsen's conception of ventriculocisternostomy for the treatment of obstructive hydrocephalus has earned a valued place in the neurosurgeon's armamentarium. It is commonly performed by leading a catheter from the lateral ventricle downward along a subgaleal or subperiosteal tract to the cerebellar dura mater and thence into the cisterna magna. Occasional complications defeat the purpose of the operation. These include necrosis and, sometimes, more or less extensive infection in the integuments of the tube, obstruction of the tube by kinking over the edge of the trephine opening, leakage of fluid through the cerebellar dura mater around the catheter, with development of false meningoceles, and migration of the tube in one direction or the other. In attempts to avoid these hazards, we gradually developed the principle of confining the tube to the subdural and subarachnoid spaces.

TECHNIQUE

We operate with the patient seated, but any position may be used if the neck is flexed adequately. A midline incision is made from inion to 3rd cervical spinous process, and the upper end is curved laterally for 3 or 4 cm. A midline suboccipital craniectomy is performed and the cranial removal is continued upward on one side under the muscular attachment to uncover the lower edge of the transverse sinus for 3 or 4 cm. If a cannula was been left in the lateral ventricle, it is removed or occluded at this stage. The dura mater is incised in the form of a U, the bend of which encroaches slightly on the cervical dura mater. The resulting flap is turned upward. The upper edge of the cerebellum is separated from the sinus well lateral to the midline and is depressed gradually to expose the inferior surface of the tentorium. An avascular-appearing area, 1 to 2 cm. anterior to the edge of the sinus, is opened with a crucial incision, exposing the inferior surface of the cerebrum. This is cauterized and is nicked with a pointed knife. A cannula is passed forward and upward into the floor of the lateral ventricle. The distance from tentorium to ventricle is noted. A #10 French rubber catheter is provided with one or two accessory openings near its tip. It is marked below its lowest opening with the distance between tentorium and ventricle by a silver clip temporarily closed upon it. The brain cannula then is withdrawn and the catheter is passed forward along its tract into the ventricle. This usually is not difficult to do. Should the catheter not follow the tract easily on the first passage, it may be stiffened by putting a 22-gage spinal puncture needle through its wall 5–6 cm. behind the tip and advancing this up the lumen to serve as a stilette. The needle is removed as the catheter enters the ventricle. The catheter is advanced until the silver clip reaches the tentorium. The clip is removed and the catheter is passed 3–4 cm. further into the ventricle. The catheter is aspirated and irrigated to clear it of detritus. Excessive drainage of fluid may be prevented by closing the tube temporarily with a silver clip.

We consider it important to anchor the distal tip of the catheter within the cisterna magna. The tube is measured down to a point slightly below the bottom of the U flap of dura mater.

It is cut off obliquely here with the opening slanting inward, to present the largest possible aperture to the cistern. No accessory openings are made in this end of the tube for fear of one becoming plugged by granulations. A mattress stitch is passed through dura mater, arach-

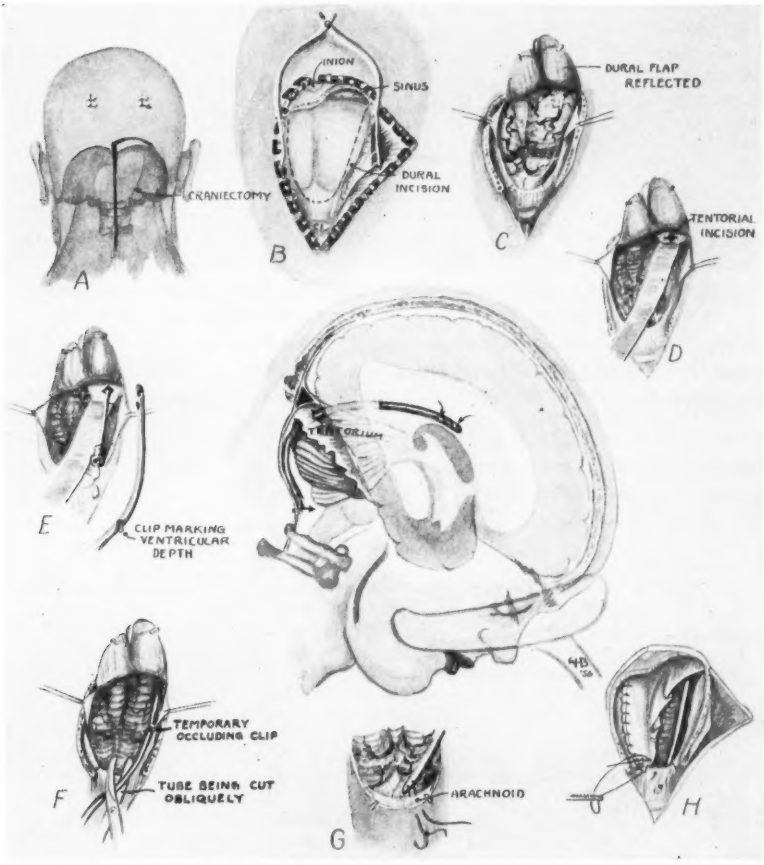


FIG. 1. Center: Scheme of transtentorial, subarachnoid course of catheter. (A) Line of incision and craniectomy. (B) Dural exposure and outline of dural incision. (C) Exposure of cerebellum and cisterna magna. (D) Exposure of tentorium by depression of cerebellum; tentorial incision. (E) Ventricle tapped with brain cannula; catheter ready for insertion. (F) Catheter, closed by clip, being cut obliquely below level of dural incision. (G) Mattress suture securing catheter within cistern. (H) Catheter in place; dural closure.

noid, catheter, and back through the same layers, to be tied outside the dura mater. This insures permanent, subarachnoid fixation of the end of the tube.

The dural flap now is replaced and sutured as water-tight as possible. We do this with a continuous locked cotton suture on a tiny staphylorrhaphy needle held in a tonsil hemostat.

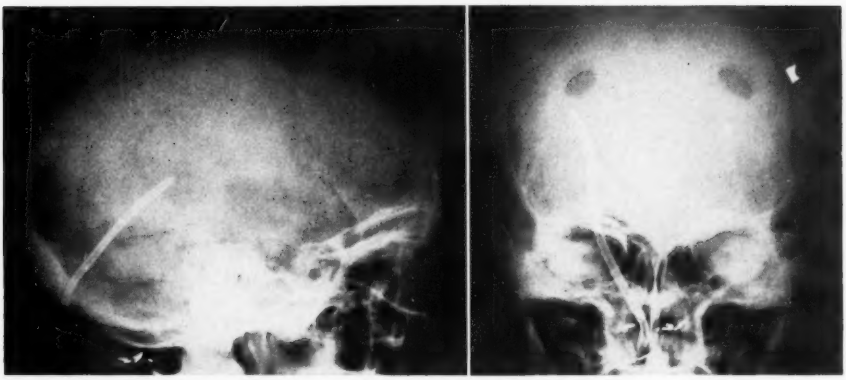


FIG. 2. Lateral and anteroposterior roentgenograms showing catheter extending from lateral ventricle, through tentorium, to end in cisterna magna.

The closed dura mater then is covered with thin sheets of Gelfoam® soaked with blood from the muscle flap. After this has adhered, the wound is closed in the conventional fashion.

These wounds do not leak, nor do the tubes migrate. The cerebrospinal fluid does not become contaminated with blood and products of necrosis from the epidural tissues. The success of the procedure has been enhanced by the adoption of this method of performance.

NOTICES

AMERICAN BOARD OF NEUROLOGICAL SURGERY

The American Board of Neurological Surgery held its Spring meeting in St. Louis, Missouri on May 18–21, 1957. There was considerable discussion at this meeting regarding a change in the training program which would abolish the inequity at present caused by the fact that certain states require a rotating internship as prerequisite to the practice of medicine. Since, however, any change in the training program would necessitate a change in the By-Laws, and since a change in the By-Laws cannot be accomplished until the proposed change has been discussed at one meeting, acted upon at the next, no definite action could be taken. A decision regarding this matter will be arrived at at the Fall meeting of the Board, and Chiefs of Services will be apprised of any change.

Examinations were given by the American Board of Neurological Surgery on May 19–21, 1957 at the Barnes Hospital, St. Louis, and the following were certified:

BELL, ROBERT L. 1351 New York Ave., Brooklyn 3, N. Y.
BIRD, CORNELIUS A. 2706 St. John Ave., Jacksonville, Fla.
CARLIN, MAURICE P. 1120 Montgomery Dr., Santa Rosa, Calif.
CARTER, WILLIAM B. 300 Electric Bldg., Watertown, N. Y.
CORRELL, JAMES W. 700 West 168th St., New York, N. Y.
FARLEY, ALBERT W., JR. 1803 North Michigan, Saginaw, Mich.
GOLDEN, JAMES B. 300 Homer Ave., Palo Alto, Calif.
GOMEZ, FRANCISCO (Foreign). Henry Ford Hospital, Detroit 2, Mich.
HANZEL, SAM 600 Mission Ave., San Rafael, Calif.
IMLER, ROBERT L., JR. 107 Utica Square Medical Bldg., Tulsa, Okla.
JOLLEY, FLEMING L. Emory University Clinic Bldg., Emory University, Ga.
LIN, PAUL M. Albert Einstein Medical Center, Northern Division, Philadelphia, Pa.
MCALHANY, HOWARD J. 401 Professional Bldg., Springfield, Mo.
MENDELSOHN, ROBERT A. Lackland Air Force Base, San Antonio, Texas
NEWSOM, WILLIAM A. 450 Sutter St., San Francisco, Calif.
SCOTT, THOMAS E., JR. 135 Broadway, Daytona Beach, Fla.
STAINSBY, DONALD L. 490 East Broadway, Eugene, Ore.
SULLIVAN, EDWARD J. Marshall Taylor Doctors Bldg., 836 Miami Rd., Jacksonville, Fla.
VAN BUREN, JOHN M. National Institute of Neurological Diseases and Blindness, National Institutes of Health, Bethesda, Md.
WILEY, J. LARUE 1079 Park Ave., Rochester 10, N. Y.
WISE, BURTON L. University of California Medical Center, San Francisco 22, Calif.

SOCIETY OF NEUROSURGERY OF CHILE

On April 13, 1957, with the assistance of the heads of neurosurgery in Santiago, Valparaíso and Concepción, as well as neurosurgeons from every Division of the city, the Society of Neurosurgery of Chile was founded. Its first board of directors is as follows: President, Dr. Alfonso Asenjo, Director of Instituto de Neurocirugía, Santiago; President-Elect, Dr. Carlos Villavicencio, Head of the Electroencephalographic Department and the League against Epilepsy of the Instituto de Neurocirugía, Santiago; Secretary-Treasurer, Dr. Juan Fierro, Head of Paraplegic Rehabilitation Center of Instituto de Neurocirugía, Santiago; Directors, Dr. Mario Contreras, Head of the Neurological Surgery Division of Valparaíso and Dr. Juan

Olivares, Head of the Universidad Católica Neurological Surgery Division, Santiago.

[Until the Society issues its own Bulletin, the review of the Instituto de Neurocirugía (*Neurocirugía*) will be the official channel for publications.

MEETINGS

American Academy of Neurological Surgery, The Cloister, Sea Island, Georgia, November 11–13, 1957. Eben Alexander, Jr., Bowman Gray School of Medicine, Winston-Salem, N.C., Secretary.

Association for Research in Nervous and Mental Disease, Hotel Roosevelt, New York, N.Y., December 13–14, 1957. Rollo J. Masselink, 710 West 168th St., New York 32, N.Y., Secretary.

Congress of Neurological Surgeons, Statler Hotel, Washington, D.C., November 7–9, 1957. Philip D. Gordy, Suite B-1, Professional Bldg., Wilmington, Del., Secretary.

First International Congress of Neurosurgery, Brussels, Belgium, July 21–28, 1957. Dr. Marcel David, Hospital Ste. Anne, Paris, Secretary-General, and Dr. William B. Scoville, 85 Jefferson St., Hartford, Conn., Assistant Secretary-General.

Harvey Cushing Society, Mayflower Hotel, Washington, D.C., April 21–23, 1958. David L. Reeves, 316 West Junipero St., Santa Barbara, Calif., Secretary.

Latin-American Congress of Neurosurgery, University of Antioquia, Medellín, Colombia, July 8–11, 1957. Dr. Ernesto Bustamante, Facultad de Medecina, Medellín, Colombia, Secretary.

Neurosurgical Society of America, Key Biscayne, Florida, January 15–18, 1958. Dr. Frank P. Smith, 260 Crittenden Blvd., Rochester 20, N.Y., Secretary.

Pan-Pacific Surgical Association, Honolulu, Hawaii, November 14–22, 1957. Dr. F. J. Pinkerton, Room 230, Young Building, Honolulu, Hawaii, Director General.

JOURNAL OF NEUROSURGERY

The *Journal of Neurosurgery* is devoted to the publication of work relating primarily to neurosurgery, including studies in clinical neurophysiology, organic neurology, ophthalmology, radiology and pathology. Short articles on unusual cases and on technique, special instruments or equipment that might be useful to others in the field are also acceptable.

MANUSCRIPTS are accepted with the understanding that they are contributed solely for publication in this *Journal.* They should be addressed to Dr. Louise Eisenhardt, 333 Cedar Street, New Haven 11, Connecticut, U.S.A. They should be typewritten in double space, with tabular matter and passages for small print on separate sheets. All materials should be mailed flat.

REFERENCES should be arranged alphabetically and cited by superior numerals in the text. The following order should be used:

Articles: Name of author, full title of article, name of periodical, year, volume, first and last page of article. Abbreviations of names of periodicals should conform to the style used in *World list of scientific periodicals published in the years 1900–1950* (London: Butterworths Scientific Publications, 1952).

Books: Name of author, full title of book, city, publisher, year, edition, full pagination (if book is in more than one volume, give the number of volumes).

ILLUSTRATIONS. Care should be taken not to use too many illustrations. All figures approved by the Editorial Board are provided free of charge with the exception of color plates which are inserted at the expense of the contributor. Permission must be obtained from the subject for reproduction of identifiable photographs of patients. If illustrations are larger than 8.5×11 inches, reduced glossy prints should be submitted instead. Any lettering should be large enough to permit necessary reduction. Paper clips should not be used on prints. A hard pencil should not be used in writing on the back of prints. Illustrations should be numbered and the top plainly indicated. Illustrations should be submitted UNMOUNTED.

SPECIAL TYPOGRAPHY. Tables and also text set up in small print in excess of 10 per cent of composition costs are charged to the author.

PROOF CORRECTIONS will be allowed without charge up to 5 per cent of the original cost of composition. The cost of changes in page proof, if made by the author, will be borne by him. *Page proof* will be submitted to authors only when special questions arise.

REPRINTS. The *Journal* supplies 25 reprints without charge to the senior author of each article. Additional reprints may be purchased according to a schedule of prices that accompanies galley proof.

SUBSCRIPTIONS. The subscription rate for the volume of six bi-monthly issues is $11.00 in the United States and Pan-American Union; $11.50 in Canada; $12.00 in other countries. Single copies are $2.00 postpaid. Single copies of Volume VIII, Number 1, January 1951 issue: A Symposium on Intracranial Vascular Abnormalities: $3.00 postpaid, if available. Volume I, Number 1 is supplied only on orders for the complete volume. Payments may be made in advance to Charles C Thomas Publisher, 301–327 East Lawrence Avenue, Springfield, Illinois, U.S.A.

CONTENTS

1. Spatial Relationships between the Globus Pallidus and the Anterior Commissure. Edgar M. Housepian, and Malcolm B. Carpenter 363
2. Laminagraphy with Cerebral Pneumography. R. A. Manfredi, and F. Kruse, Jr. 374
3. Polarographic Study of Cortical Blood Flow in Man. John S. Meyer, and John Hunter 382
4. Phrenicofacial Nerve Anastomosis for Facial Paralysis. Robert C. Hardy, George Perret, and Russell Meyers 400
5. A New Technique for the Microscopic Examination of Cerebral Vessels *in Vivo*. Salamon Hakim, and C. Miller Fisher 405
6. The Use of Radioactive Iodinated Serum Albumin in the Localization of Intracranial Lesions. Richard B. Rhody, and Gerald R. Nowlis 413
7. Graphic Interpretation of Isotope Localization of Intracranial Lesions. Shelley N. Chou, and Lyle A. French 421
8. The Effect of Carotid Arteriography, Using 35 Per Cent Diodrast, on the Protein Content of the Spinal Fluid. George T. Tindall 430
9. Prognosis of Subarachnoid Hemorrhage. A Comparison between Patients with Verified Aneurysms and Patients with Normal Angiograms. G. af Björkesten, and Henry Troupp 434

Special Article:

The Clinical Use of Electroencephalography. Paul C. Bucy 442

Case Reports and Technical Notes:

Bilateral Symmetrical Aneurysms of Internal Carotid Artery within the Cavernous Sinus. Case Report. Jacob Seltzer, and Everett F. Hurteau 448
Partial Glossopharyngeal Neuralgia Associated with Syncope. Hendrik J. Svien, Norman C. Hill, and David D. Daly 452
Brain Abscess in Two Patients with Congenital Cardiac Disease. E. Ley, S. Obrador, and E. Ley, Jr. 458
Multiple Primary Neoplasms of the Central Nervous System. Report of a Case of Spinal Ependymoma and an Intracranial Meningioma. Interval of Fourteen Years. James Greenwood, Jr., and T. H. McGuire 462
Brachial Vertebral Angiography in Adults. William F. Collins, Jr., Harry W. Slade, and William G. Lockhart 466
New Instrument. Frank H. Mayfield 469
A Modification of Torkildsen's Ventriculocisternostomy. Carl J. Graf, and Wallace B. Hamby 470
Notices 473

GEORGE BANTA COMPANY, INC., MENASHA, WISCONSIN, U.S.A.